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A TREATISE

ON THE

DISEASES OF THE HEART

AND

GREAT VESSELS.

A

TREATISE

ON THE

DISEASES OF THE HEART

AND

GREAT VESSELS,

COMPRISING A

New View of the Physiology of the Heart's Action.

ACCORDING TO WHICH THE PHYSICAL SIGNS ARE EXPLAINED.

By J. HOPE, M.D.

SENIOR PHYSICIAN TO THE ST. MARY-LE-BONE PAROCHIAL INFIRMARY:
FORMERLY HOUSE PHYSICIAN AND HOUSE SURGEON TO THE ROYAL INFIRMARY
OF EDINBURGH: EXTRAORDINARY MEMBER, AND FORMERLY PRESIDENT,
OF THE ROYAL MEDICAL SOCIETY OF EDINBURGH: MEMBER OF THE
SOCIÉTÉ D'INSTRUCTION MÉDICALE OF PARIS, ETC.

> "Most of the phenomena which nature presents are very complicated; and when the effects of all known causes are estimated with exactness, and subducted, the residual facts are constantly appearing in the form of phenomena altogether new, and leading to the most important conclusions." HERSCHEL, Prelim. Disc. p. 156.

LONDON:
WILLIAM KIDD, 228, REGENT STREET;
ADAM BLACK, EDINBURGH; AND T. ATKINSON AND CO. GLASGOW.

M.DCCC.XXXII.

540,

PREFACE.

I CANNOT dismiss this work without expressing my great obligations to the gentlemen mentioned pages 11, 12 and 15, for the able assistance which they afforded me in the performance and attestation of the experiments.

To Dr. Forbes of Chichester, Dr. Charles Williams, and Dr. G. Julius of Richmond, whose intimate knowledge of diseases of the chest and of auscultation, requires no panegyric from me, I am likewise indebted for many valuable hints and suggestions in the plan and execution of the work.

To Dr. Lombard of Geneva, I have also to express my acknowledgments for the two remarkable cases xxx and xxxi.

J. H.

13, *Lower Seymour Street, Portman Square,*
Nov. 25, 1831.

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INTRODUCTION.

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Preceded by names so distinguished as those of Corvisart, Kreysig, Burns, Laennec, and Bertin, I am sensible that I expose myself to the imputation of presumption, in offering to the profession a new treatise on the diseases of the heart and great vessels. I feel called upon, therefore, to explain, in a more circumstantial manner than I should otherwise have wished, the motives which have induced me to undertake this work, and the plan which I have pursued in its execution. Whether I am justified in the attempt I can scarcely form an opinion. Every author contrives, I believe, to persuade himself that the work which consumes his own midnight oil, is precisely the one that is wanted. It is for the reader to determine whether I labour under the delusion common to my brethren.

Notwithstanding the strong light diffused over the diseases of the heart by the researches of the above-mentioned authors,—notwithstanding the brilliant sunshine emanating from the discovery of auscultation by Laennec,—a discovery, which, according to M. Bertin, "has, in a few years, more completely illumined the diagnosis of the diseases in question, than all the other modes of exploration had done for two centuries;" the great body of the profession still deny that the piercing ray has reached its destination, still doubt the utility of auscultation in reference to the primary organ of the circulation, still find the ordinary symptoms beset with their accustomed difficulties, still complain, in short, that the obscurity which involves the diseases of which we speak, is scarcely less profound than ever:* and, while conflicting opinions are embarrassing the judgment, and undermining the confidence of the patient investigator of truth, there is a general outcry for an additional mass of well attested evidence, which may bring the subject to some kind of a conclusion.

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* A distinguished Frenchman recently said to me, "Monsieur, je ne crois pas, pour vous dire la vérité, que l'on puisse en faire le diagnostic—que sur la table du salon."

It rarely happens that a general impression is wholly unfounded: nor is it, if I mistake not, in the present instance. Authors actually have not succeeded in completely redeeming this subject from its obscurity. Errors remained to be corrected, deficiencies to be supplied, inconsistencies to be reconciled: the subject—a confused and incongruous mass, required to be moulded and compacted into a symmetrical and harmonious whole, the parts of which, though perfect in themselves, should, by their justness of proportion and unity of design, afford relief and support to each other.

I proceed to glance briefly at the subjects where the principal defects appear to have resided; and this I do, not only for the purpose of general guidance to the student, but also for that of pointing out where I have differed from preceding writers. In these differences, I am anxious to offer my opinions, not as established facts, though I trust that they will be found grounded on careful observation, but simply as propositions to be admitted or rejected according to the test of general experience. I am satisfied that, in our profession more especially, where there are few *fixed* points to constitute the basis of an inductive process, nothing is more difficult to ascertain than a *general* fact. Innovations, therefore, cannot be regarded with too much suspicion, cannot be scrutinized with too much severity, cannot be received with too much caution and reserve.

The most prominent error which reigns throughout the doctrines of Laennec, and which has prevailed in the schools since the first publication of his work, is, that he mistook the nature of the action of the heart. I trust that the view which I have ventured to substitute may be found more satisfactory; and, as nearly a year and a half has elapsed since I first published my experiments and clinical observations relative to it: as my conclusions have, throughout that period, remained, so far as I can judge, uninvalidated; and as I have recently repeated the experiments with the same results, before a number of the most distinguished physiologists and pathologists of the metropolis;* I hope I shall not be considered precipitate in having taken the decisive step of modifying and explaining all the physical signs of disease of the heart according to the view in question.

Laennec and his predecessors have assigned to diseases of the heart a certain series of symptoms, which they conceived to be common to the whole; but they had not analysed those symptoms and ascertained which were peculiar to, and pathognomic of, the several affections taken individually. MM. Bertin and Bouillaud, both writers of high talent, made this attempt, and with partial success; but the spirit of generalization, (if I am correct in my own views,) carried them a grade too far. What observation leads me to regard as an inaccuracy, constitutes the hinge of their work—the pivot on which turns the principal train of their reasoning: namely, that the symptoms of a retarded circulation are, under all circumstances, the result of a *mechanical obstacle* to the course of the blood:—that when, for instance, they accompany hypertrophy or dilatation, they are not consequences of these affections, but of some co-existent mechanical obstacle, as a contracted valve, an aortic aneurism, &c. I have attempted to show, not only that hypertrophy and dilatation can, of themselves, respectively occasion the symptoms in question; but, that these symptoms are seldom produced in any very remarkable degree of severity by a mechanical obstacle, unless hypertrophy, dilatation, or softening of the heart is superadded.

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* Vid. p. 11, 12, 15.

It may naturally be supposed that the erroneous view which Laennec took of the heart's action, led to corresponding errors in his doctrines of auscultation. Yet these are, fortunately, not of such magnitude as might be expected. With one exception—that of considering loudness of the second sound to be an indication of dilatation of the auricles, the errors are those of omission, and of incorrect explanation. The omissions are considerable and important. He was not aware of a fact noticed by the writer several years ago, that bellows-murmurs are produced by regurgitation through the valves. This oversight alone naturally shook the confidence of many, and eventually of himself, in his theory of valvular murmurs. For, the lesion being found in one valve, when, according to that theory, it was expected in another, the inevitable conclusion was, that the theory was incorrect. At the same time, the cause of the murmur remained doubtful.

The perplexity was further increased by the existence of murmurs independent of valvular disease, and accompanying both hypertrophy with dilatation, and a nervous action of the heart without any organic lesion whatever. These murmurs Laennec attributed to a wrong cause: viz. to the sound of the muscular contraction, instead of to the modified motion of the fluid; which I presume to consider the true cause. Hence, he was unable to analyse and foresee the circumstances under which nervous murmurs should occur, and, consequently, to distinguish them from those occasioned by valvular disease.

Several minor phenomena likewise, as the purring tremor, and the arterial thrill and bellows-murmur, he was, in consequence of the confusion created by the error in question, equally unable to explain. Hence, he vaguely attributed them to some unknown "modification of the nervous action."

It cannot be a subject of surprise that, with the above opinions, acquired chiefly during the latter period of his life, he should have retracted, in his second edition, the much more accurate doctrines respecting bellows-murmur as a sign of valvular disease, which he had advanced in his first;—transmitting to his disciples the confusion which reigned in his own mind, but which, like the storm that, in tropic climes, is the precursor of the purest, brightest weather, must, sooner or later, had his life been spared, have rolled away before the irresistible force of his purifying and enlightening genius.

The murmurs attending valvular disease, hypertrophy with dilatation, nervous action of the heart, reaction from loss of blood, pericarditis, and adhesion of the pericardium; also the allied phenomena of purring tremor, arterial thrill, throb, and murmur, I have attributed to modifications in the motion of the blood, and explained according to the laws of hydraulics and acoustics. In this way, not only may organic diseases of the heart be readily and certainly distinguished from nervous and other affections wearing their aspect, but, with attention to certain rules which I have offered respecting the situations where valvular sounds are to be explored, and to certain corroborations derived from general symptoms, the particular valve diseased may in general be detected with precision. Such, at least, are the conclusions to which I have been brought by a very considerable number of cases, a small proportion of which are appended to this volume.

The investigations of Laennec on aneurism of the aorta, were limited and inconclusive: accordingly, he remarks that, "of all the severe lesions of the thoracic organs, three alone remain without pathognomonic signs to a practitioner expert in auscultation and percussion,—namely, aneurism of the aorta, pericarditis, and polypi in the heart previous to death." I hope that my attempts to throw light on these subjects, may not be found entirely fruitless. The article on aneurism is the substance, with considerable additions, of a series of essays published in the Lond. Med. Gaz. Aug. 22, 1829, and is founded on nearly forty cases in which the diagnosis was verified by post-mortem examination.

The treatment of diseases of the heart offers a spacious field for improvement. Previous to the discovery of auscultation, these maladies could seldom be detected before they were so far advanced as to be incurable; and then was not the time to judge of the efficacy of remedies. Laennec, absorbed in his investigation of the diagnosis, paid comparatively little attention to the treatment. His first edition scarcely alluded to it: in the second it is only cursorily treated. Bertin and Bouillaud are not more satisfactory,—giving a bold outline of leading principles, such as might be struck out by generalization in the closet, but seldom descending into those detailed delineations of therapeutic measures, which are essential to the practitioner at the bedside.

Nor are these principles always, perhaps, perfectly sound. Their habit of attributing the symptoms of a retarded circulation, under all circumstances, to one cause only,—a mechanical obstacle, gives a wrong bias to the mind; and that of entwining inflammation with the cause of almost every organic lesion of the heart or great vessels, is replete with danger to the inexperienced practitioner. While I feel bound to say this, (for it is the duty of a writer to point out the path which is insecure, no less than that which is safe,) let me not be supposed to detract from the singular merits of these authors: let me offer my tribute of admiration to the talent which shines through every page of their elegant and scientific work, and acknowledge the extensive obligations that I owe it in the execution of my own.

Conscious of the gap that was presented in the treatment of diseases of the heart, I have devoted more attention to this, than to any other department of the subject: availing myself, in particular, of the wide and favourable sphere for observation, afforded by a long residence as House Physician and Surgeon successively, in the Royal Infirmary of Edinburgh; where living, literally, I may say, as well as figuratively, at the bed-side of the patient, I had an opportunity of closely watching every habitude and phasis of the disease—every operation and effect of remedies. The result of these researches were submitted in a memoire to the Royal Medical Society of Edinburgh, in the year 1824-5.

Many think that the expectation of effecting an improvement in the treatment of diseases of the heart, is chimerical: and they think so because, not being accustomed to recognise the diseases in question before they have attained an advanced stage, they are pre-occupied with the old and popular idea of their incurability. To such it might, perhaps, be a sufficiently philosophical answer to reply, that an improved knowledge of the nature and causes of a disease, must alone necessarily lead to an improvement in the treatment; and that therapeutic weapons are dangerous when wielded in the dark. But here we may go much farther: we may say that, by the improved means of diagnosis, the maladies under consideration may be recognised, not only in their advanced, but in their incipient stages, and even when so slight as to constitute little more than a tendency. We may say on the grounds of incontestible experience, that, in their early stages, they are, in a large proportion of instances, susceptible of a perfect cure; and that, when not, they may, in general, be so far counteracted as not materially, and sometimes not at all, to curtail the existence of the patient. We may, accordingly, predict that the term "disease of the heart," which at present sounds like a death-knell when uttered by the physician, will hereafter become by familiarity not more alarming than the term *asthma*, under which it is frequently disguised.

Such are the *direct* practical improvements to be expected from a better knowledge of diseases of the heart. But there are collateral ones of no less magnitude. It has been stated by M. Richeraud, repeated by Bertin, and echoed by all who are conversant with this class of maladies, that "hypertrophic enlargement of the heart is more closely allied to apoplexy and palsy than the apoplectic constitution itself.*

Should the hypertrophy be recognised, its effects on the brain may be counteracted by judicious treatment: should it be overlooked the patient, with a view to reducing his *apoplectic fulness of habit*, is ordered smart exercise, which, by increasing the action of the heart, already too powerful, causes a preternatural determination of blood to the brain and induces the apoplectic or paralytic seizure. According to evidence hereafter to be adduced, the majority of those who are prematurely cut off by apoplexy in the apparent enjoyment of good health, sink under the circumstances described.

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* This constitution consists, according to the popular idea, in a broad, robust frame, full habit, and florid complexion. It is in general attended with an unusual size and thickness of the heart.

Again, there are few more common and certain exciting causes of palpitation and difficulty of breathing in disease of the heart, than derangement of the stomach. What happens to the patient in this case? Tracing the attack, in perhaps every instance, to a dyspeptic fit, he naturally concludes that the latter is the cause: that it is "all indigestion." "Good air, and plenty of exercise" are the remedies recommended: the result is an apoplectic seizure. The circumstance that before the introduction of the new mode of exploring diseases of the heart, they could rarely be detected in their early stages, contributed to the error in question. For, as patients frequently recover from the early stages, the recovery was regarded by those who assumed this class of diseases to be incurable, as a proof that the affection was merely dyspeptic. Hence dyspepsia acquired the reputation of producing certain symptoms, particularly in the head, which are in reality foreign to it, being exclusively the results of a co-existent disease of the heart.

There prevails another error, the converse of the above—that of mistaking nervous, or really dyspeptic palpitation, for disease of the heart. The frequency of cases of this kind, especially amongst men of studious habits, (and more particularly, I have noticed, among those of my own profession,) is truly surprising; and, as it has always been considered difficult, and by many impossible, to distinguish the two affections, the alarm created is sometimes distressing. Having thought this subject of so much importance as to demand a separate article, I shall here only say, that, so far as my own experience enables me to judge, the discrimination may be made with ease and certainty.

An immense proportion of asthmas—and of the most dangerous and distressing cases, result from disease of the heart: the same may be said of dropsies, especially those that are universal. If the cause be overlooked, the asthmatic is harassed with a farrago of inappropriate and unavailing, not to say pernicious remedies; and the hydropic is treated with dangerous activity; or for imaginary affections of the liver, the lungs, or the kidneys. On the other hand, if the cause be detected in the incipient stage, by precautionary measures both the one effect and the other may in general be prevented.

In acute rheumatism, there is no more common and formidable source of danger than inflammation of the heart and its investing membranes. Should it be overlooked when existing in a severe form, (and even in that form it is one of the most obscure and insidious of maladies,) the patient almost invariably dies from the immediate effects of the attack, or becomes a short lived martyr to an incurable organic disease of the heart.

There is scarcely a disease of the heart, accompanied with obstruction of the circulation for any considerable period, which is not productive of enlargement of the liver, and, sooner or later, of its ordinary consequence, abdominal dropsy. Yet there are few common facts in medical science less generally known than this intimate connection between the heart and the liver. The dropsy is ascribed to the latter; the treatment extends not beyond this organ; the unknown cause continues to reproduce its effect, and the patient, if he obtain relief at all, only obtains it to undergo a speedy relapse.

Individuals affected with disease of the heart are peculiarly liable to inflammation of the lungs; and such inflammation, as I have endeavoured strongly to inculcate throughout this volume, is singularly rapid and destructive. Yet if, from ignorance of the state of the heart, free depletion be practised on the ordinary principles, the patient may sink suddenly after the first or second abstraction of blood. I have more than once witnessed this catastrophe, and few practitioners of experience have not seen the same.

In fever and inflammation in general, disease of the heart may impart to the pulse dangerously deceptive characters of hardness, fulness, weakness, or irregularity, and the patient may be bled too much, from the prevalence of the former characters, or too little, from the presence of the latter.

Thus it is seen that the practical improvements to be derived from a better knowledge of the diseases of the heart, extend, not to the diseases of this organ alone, but to a multitude of the most formidable maladies incident to the human frame. There is, in short, scarcely an affection with which disease of the heart may not be more or less interwoven; and, "if," to use the language of Senac, "we would not pronounce rashly on an infinity of cases; if we would not harass our patients by noxious and unavailing remedies; if we would not accelerate death by treating certain diseases like others which are entirely different; nor be exposed to the disgrace of seeing our diagnosis falsified by the results of dissection; finally, if we would not have danger to be imminent, whilst we are under the blind impression that it is remote, we must study the diseases of the heart."

Such appear to be the vacuities left by preceding writers, and such the advantages to be anticipated from their being supplied. It remains for me to explain the plan of the present work and glance at a few particulars in its execution.

The work is divided into six parts; I. The Anatomy and Physiology. II. Inflammatory affections. III. Organic affections. IV. Nervous affections. V. Miscellaneous affections. VI. Cases. Although every arrangement of diseases of the heart presents considerable difficulties, and I am by no means perfectly satisfied with the one which I have adopted, it appears to me preferable to others, because affections of the same class, being thrown together, by juxta-position reflect light upon each other; nor, at the same time, are the inflammatory, and the organic affections in general so intimately connected, as to render their separation impossible without doing violence to the continuity of the subject. The miscellaneous affections are ranged by themselves, because they are not reducible to any of the preceding heads.

In the execution of the work, it has constantly been my aim, by studying the symptoms in connexion with the morbid anatomy, to trace the alliance of the two as cause and effect, and thus to reduce them to certain general and intelligible principles, which might not only contribute to future accuracy of observation, but facilitate the registration of so many and so complicated facts in the memory. I have studied of late years more particularly to accomplish this object, in consequence of having witnessed the complete success of a similar attempt by Dr. Charles Williams, in his "Rational Exposition of the Physical Signs of Diseases of the Lungs;" beyond comparison the best original work that has appeared on auscultation since that of its discoverer, and which ought to be the first book in the hands of every one who wishes to arrive at a sound knowledge of stethoscopy by the most expeditious route.

As the authenticity of cases and observations is of the first importance, I deem it necessary to present a short explanation of the manner in which I have conducted my investigations. Being persuaded that no evidence is so suspicious as that of the senses, because the magnitude of an error is in proportion to the certitude which is supposed to attach to that mode of exploration, it has constantly been my endeavour to avail myself of the collective testimony of many. Accordingly, I have, for publication, preferred hospital cases, as being the best attested; I have invariably *written* the opinions or *diagnoses* before the death of the patient; have publicly tested them by the results of post-mortem examination; have minuted the dissections with the subject before me, and according to the prevailing opinion of the individuals present; and, generally before laying down my journal, I have annexed such remarks as the case suggested, while the circumstances were fresh in my recollection. Finally, I have obtained signatures where a case was very remarkable, or where there appeared a possibility of its being subsequently called in question. The cases appended to this work are nearly verbatim transcripts from journals thus kept; and, in order that they might present a just idea of the possibility of detecting disease of the heart, I have not taken them by selection, but, excepting a few, mostly without diagnoses, have introduced the whole of which I took notes in St. George's Hospital within a definite period. They will be found, I believe, to substantiate the view which I have offered of the heart's action—according to which the physical signs are explained; and, to the practical student of auscultation, by standing in the relation of exercises to a grammar, I entertain hopes that they may prove one of the most acceptable portions of the volume.

The hospital researches alluded to have been conducted at the Royal Infirmary of Edinburgh, as above stated: at St. Bartholomew's, London: at La Charité, Paris, where the lessons and researches of MM. Chomel,* Andral, and Louis, afforded the most favourable opportunities for studying auscultation: at the Santo Spirito, Rome: and, finally, at the Mary-le-Bone Infirmary and St. George's Hospital, London. From these and private sources I have minuted a greater number of cases, than has, I believe, been published by any previous author.

In some parts, I have occasionally introduced repetitions. Thus, in describing the mode in which changes of structure produce their pathological effects, I have glanced at the symptoms; and in describing the symptoms, I have explained them, where practicable, by the changes of structure. This I have done designedly; for I am satisfied that such is the process of thought which passes through the mind at the bed side and in the post-mortem theatre; and a practical work ought to be the transcript of the mind in those two situations. I have, likewise, made occasional repetitions in the treatment with the view of saving the reader the inconvenience of frequent reference.

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* I owe it to the politeness of the French nation in general, and of this gentleman in particular, to state, that he not only granted me the privilege of being one of his clinical assistants; but, as I was engaged in making drawings of morbid structure, he also allowed me the immediate use of the best specimens which his wards afforded, purposely postponing the demonstration of them to his class till the following morning.

Wherever the subject was one of original research, or otherwise particularly important, I have been circumstantial. Aneurism of the aorta, hypertrophy, the signs of disease of the valves, &c. may be cited as instances. Where the subject was known, I have presented those points only, of which I am myself conscious of making use in practice, suppressing many subordinate minutiæ, which, though essential to original researches, gradually become superfluous, in proportion as the alchemic process of generalization assays, and assigns their full value to leading facts. Accordingly, I must refer the reader to Laennec for many details, which evince the astonishing accuracy and extent of his first researches, but which are no longer requisite for practical purposes. On the subject of the morbid anatomy of the heart I have been minute,—perhaps tediously so; but it has appeared to me necessary, because there is perhaps no organ in the body, of the diseased states of which the generality are less competent judges than of the heart; and this is the source of the frequent and dangerous error of confounding organic with nervous disease, or of overlooking the former entirely.

I am prepared to expect some dissent from my views respecting asthma as symptomatic of disease of the heart. Being the results of observation, I submit them with confidence, but shall be the first to recant, should they be demonstrated to be erroneous. I learn that M. Rostan entertains similar views, but, having completed my manuscript, and thinking nature a sufficient guide, I have refrained from consulting his works.

With respect to the comparative value of the general and physical signs of disease of the heart, it may be said that Laennec rather undervalued the former and over-rated the latter. This was owing principally to the general signs being less perfectly understood when he studied than they have subsequently become in consequence of being investigated with the aid of auscultation. The ardour of his early disciples, who imagined that the physical, rendered the general signs superfluous, brought auscultation into some disrepute by the inaccuracy of their diagnosis. But since the stethoscope has taken its proper place as an auxiliary only, and the diagnosis has been founded on the two classes of signs conjointly, auscultation has ranked as a discovery which will immortalize its author and form an epoch in the history of medicine.

	Right Ventricle.		Right Auricle.		Left Ventricle.	
	Hypertrop.	Dilatation.	Hypertrop.	Dilatation.	Hypertrophy.	Dilatati
I. Briant, Robert,	Considerable.	Dilatation.			Immense.	Dilata tion.
II. Andrews, Mary					Simple hypertrophy.	
III. Collard, Richard	Rather less than on left.	Rather less than on left.			To ¾ of an inch thick.	Great, lemo size.
IV. Keith, David	Natural, about.	To half larger.			To one inch.	To hal large than nat ral.
V. Green, John	Natural.	To twice.			¾ inch thick.	To twi
VI. Copas, John	Less than on left side.	Less than on left side.			1½ inch.	Enorm larges orange
VII. Snowden, John	About natural, or rather thin.	Less than left.			Nat. ½ inch: a polypus containing pus.	Conside able.
VIII. Gillan, Patrick	Natural or rather thin.	To double.			Natural or rather thin.	Near double
IX. Quin, Maria		Slight.	Doubtful.	Doubtful.	Thin rather.	
X. Lambert, William	Five lines thick.	Less than on left.	Dilated.	Dilated.	Extreme attenuation one line.	Enorm 3 or 4 times.
XI. McEarl, Henry	Slight.	Half larger.			¾ inch thick.	Half larger.
XII. Porter, Richard		Dilated.			One inch nearly.	Dilate
XIII. Hedgley, William	Slight.				Above ½ inch though only æt. 10, and very firm.	
XIV. Storer, Richard		Dilated.		Dilated.	Hypertrophy if either.	Dilate
XV. Dolan, John		Dilated.		Dilated.		Dilate

S

n.	Lef Hypert		Pulmonary. Apoplexy.	 Œdema. Emphysema.	Murmurs.
.		and ar.		Emphysema.	
		be- syst.			Loud rasping murmur.
a		ng, ing.		Œdema of lungs.	Whizzing in aorta.
lf tu-		reg. last qual		Emphysema and œdema; hepatization of lower lobes.	
e.		full.		Emphysema and œdema; hepatization.	
l. t		rong lar.			Not explored.
r-		reg^r. ding r se- nd.			
		120, very nt.			
	Doubt	soft ll.			
	Dilate	and very nt.	Pulmon. apoplexy.		
		o80, , str. ting.			Murmur from aorta.
l.		nd irm. r.			Bruit de râpe very loud.
		very eak ilar.			Bruit de scie from aortic valves.
l.		full, nd	Lung condensed, bronchial dilatation.		Bellows-murmur.
l.		all eak.	Pulmon. apoplexy.		Loud. Soft, No back stroke.

ABL

pid Valve.
il. ring, f middle nger.
widened, margin, and thick closed.

PART I.

ANATOMY AND PHYSIOLOGY OF THE HEART.

CHAPTER I.

ANATOMY OF THE HEART.

A Knowledge of it essential, 1. *Active Diastole,* 2. *Size and Proportions of the Heart, absolute and relative,* 2. *Exact Situation of the Heart and great Vessels,* 3. *Situation of the Lungs over the Heart,* 5. *Situation of the Auricles,* 5. *Situation of the Pericardium,* 5. *Changes of Situation occasioned by Enlargement of the Heart and Hydro-pericardium,* 5, 6. *Percussion of the Heart,* 6. *Plessimeter,* 7. *Rationale of Resonance,* 7.

As morbid anatomy and pathology are only comparative states, or the amount of a deviation from the healthy standards of anatomy and physiology, it is essential for these standards to be thoroughly understood, before the morbid deviations can be appreciated. Of the descriptive anatomy of the heart it is not, however, my intention to treat, as this subject presents no obscurity, and as it ought to be studied in much greater detail than is consistent with the plan of the present work. I pretermit, likewise, that portion of the physiology which relates to the arrangement and action of the muscular fibres, referring the reader to Stenon, Wolff, Duncan, Gerdy, and other original sources of information. It may be briefly observed that some imagine the systole to be effected by the contraction of a certain set of fibres, and the diastole by that of another; that, in short, the latter, as well as the former, is the result of an *active* muscular effort. This, however, has not yet been satisfactorily demonstrated; and, while awaiting the issue of further research, it is perhaps safer, for the present, to attribute the diastole to that power by which a muscle reverts from the state of contraction, to that of relaxation, and which I shall, for the sake of avoiding circumlocution, designate by the title *elasticity*.

There is one point, which is generally treated in too cursory a manner by descriptive anatomists, and the thorough knowledge of which is absolutely essential to the study of diseases of the heart. I allude to the relative size of the organ to the whole frame, and of its several compartments to each other. It is ignorance in this respect that has for centuries caused thickening, attenuation, enlargement, and diminution to be overlooked, and the symptoms of disease of the heart to be attributed to any cause but the legitimate one. As the subject might escape notice if introduced in this place, I have treated of it immediately before describing the anatomical characters of hypertrophy, where it will be both conspicuous and convenient.

A knowledge of the exact situation of the heart, is a point of no less importance to the auscultator; and, though it does not strictly come under the head of anatomy, I shall, for convenience, advert to it here.

As the apex and body of the heart are free, while the base, secured by the great vessels, is comparatively, though not absolutely fixed, the organ turns in a slight degree upon its base with each alternate movement of the diaphragm, the descent of the muscle causing its longitudinal axis to assume a more vertical position, and the ascent throwing it transversely to the left. It is necessary, therefore, that the anscultator fix upon some given point at the base, which may serve as a mark and guide for his exploration of the situation of the organ. The point which to myself has appeared the most certain, is, the pulmonary artery. This vessel, midway between its origin and the place where it divaricates into the two trunks distributed to the lungs, bulges at the interspace between the second and third left ribs close to the sternum, a circumstance which, as well as the situation of the other parts of the heart, I have carefully ascertained by forcing needles through the thoracic walls, at given points, into the viscera beneath. The situation of the pulmonary artery was, also, well displayed by the dilatation of that vessel described in case xx. (Weatherly.) A line drawn from the inferior margins of the third ribs across the sternum, passes over the pulmonic valves a little to the left of the mesial line, and those of the aorta are almost directly behind them. From this point the aorta and pulmonary artery ascend; the former inclining slightly to the right, coming in contact with the sternum when it emerges from beneath the pulmonary artery, and following, or perhaps rather exceeding, the mesial line, till it forms its arch; the latter, which is, from the first, in contact with the sternum, inclining more considerably to the left, until it arrives at the interspace between the second and third ribs above described. A vertical line coinciding with the left margin of the sternum, has about one third of the heart, consisting of the upper portion of the right ventricle, on its right; and two thirds, composed of the lower portion of the right ventricle and the whole of the left, on its left. The apex beats between the cartilages of the fifth and sixth left ribs, at a point about two inches below the nipple and one inch on its sternal side.

The lungs descend along the margins of the sternum about two inches apart, and overlap the base of the heart, slightly on the right side, and more extensively on the left: then, receding from each other, they leave a considerable portion of the right ventricle, and a less extent of the lower part of the left, in immediate contact with the sternum.

The right auricle is in front of the heart, at its right side and upper part. One portion of it is overlapped by the right lung, and another, principally the appendix, is in contact with the sternum. The left auricle is situated deep behind and to the left of the heart at its upper part, opposite to the interval between the cartilages of the third and fourth ribs. The extremity of the appendix is visible in front, but, when the volume of the heart is natural, it is not in contact with the sternum, being considerably overlapped by the left lung. The pericardium ascends on the great vessels as high as the commencement of the arch of the aorta, and opposite to the second ribs.

When the heart is enlarged, its longitudinal axis becomes placed more transversely, and its lateral diameter is increased. Hence, the right ventricle projects more considerably to the right, sometimes under the whole breadth of the sternum; and the left extends far beyond its usual limits to the left, sometimes elevating by compression that portion of the lung which overlaps it, so as to bring nearly its whole surface, and the tip of the auricular appendix, in contact with the sternum. In addition to being broader and placed more transversely, the organ descends lower than natural—its apex sometimes beating between the sixth and seventh ribs, and its pulsation extending to the epigastrium.

When the right auricle is dilated or gorged, it extends upwards and to the right, and comes more extensively in contact with the sternum.

When the pericardium is distended to the utmost with fluid, it forms a pear-shaped bag, the top or narrow extremity of which sometimes mounts even above the second rib: its sides are nearly in contact with the sides of the heart, while its front is separated from the anterior surface of the heart, in the dead subject, by two or three inches of interposed fluid.

From the above description the auscultator will understand in what situations to explore the lesions of the various parts of the heart.

The percussion of the organ is so intimately connected with its anatomical relations, that I may perhaps be allowed to sacrifice strictness of arrangement to practical convenience, and advert to the subject at present.

Though percussion on the back of the fingers, firmly applied to the chest, suffices for ordinary purposes, the *plessimeter*, lined with wash-leather, to prevent its clacking, is preferable when great delicacy is required. Of this I feel assured, both from having attended the original experimental researches of M. Piorri, and from considerable subsequent experience. It is scarcely necessary to say that percussion on a solid, as the heart where it is in contact with the sternum, elicits a dead sound; while that on a body containing air, as the lungs, stomach, &c. produces a hollow sound. It is less known, and still less believed, that a solid, beneath a sonorous body, as the liver beneath the lung, the outline of the heart beneath the lung that overlaps it, &c. may be recognised by a sound intermediate between hollow and dead. Had M. Piorri, to whom this discovery is due, explained the principle of the phenomenon according to the laws of acoustics, he would at once have made it obvious that what has often been regarded as the offspring of his imagination, was the necessary consequence of an immutable law of nature. Thus, when the vibrations of the air impinge on a non-resonant or inelastic surface, they are arrested and the sound becomes deadened. This happens to the voice when exerted in a room lined with drapery. The pedal and damper of a piano are constructed on the same principle, the only difference being, that the check is given to the vibrations of the wires themselves, instead of to those of the air. Thus, when a note is struck, the vibrations continue till the finger is raised, simultaneously with which action the damper falls on the wires and arrests their movement. When the open pedal is depressed, the damper is permanently raised, and the vibrations continue whether the finger be removed or not. To apply this principle to the percussion of the chest,—sonorous vibrations excited in the lung, are arrested when they impinge upon a solid, inelastic body beneath, as the liver, heart, &c. hence the sound, at first hollow, presently becomes dead. To elicit these characters distinctly, a loud sound should be produced; and this may be effected by strong percussion, and by pressing the plessimeter firmly down, so as to condense the soft wall of the chest and render it a better conductor of sound. When there is no subjacent solid body, the sonorous vibrations expand freely and yield a proportionably hollow sound. Having just tried the experiment before several individuals placed at remote parts of a spacious room, I find that they readily distinguish the full, hollow tone of the middle lobe of the lung, the duller intonation of the lung overlapping the heart or liver, and the dead sound of the præcordial region where the heart is in contact with the chest. Accordingly, the circumference of this organ may be measured with considerable nicety by percussion on the plessimeter.

CHAPTER II.

ON THE ACTION OF THE HEART.

SECTION I.

EXPERIMENTAL RESEARCHES ON THE ACTION OF THE HEART.

Sounds of the Heart, 10. *Error of M. Laennec*, 11. *Experiments on the Rabbit*, 13, *on the Frog*, 14, *on the Ass*, 15. *First Series*, 21. *Second Series*, 30.

When the ear or a stethoscope is applied to the præcordial region, two successive sounds, followed by an interval of silence or repose, are distinctly heard. The former, which is synchronous with the impulse, and, in vessels near the heart, with the pulse, is duller and slower, terminating without an appreciable interval in the latter, which is louder and smarter, like the flapping of a bellows' valve. These sounds, first noticed by Laennec, were attributed by him the one to the ventricular, the other to the auricular contraction, and this doctrine remained unquestioned for a period of eight or ten years, until Mr. Turner, supported by the authority of the old physiologists, Haller, Harvey, Lancisi,

&c. pointed out that the auricular contraction, to which Laennec attributed the second sound, *preceded* the ventricular, and, consequently, that his theory was erroneous. Notwithstanding the talent and ingenuity displayed by Mr. Turner in proving this, he was not equally successful in assigning the cause of the second sound; and, though various theories were subsequently proposed, the nature of the heart's action remained a mystery, until it was made the subject of a series of experiments instituted by the writer in the summer of 1830, and repeated in that of 1831.*

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* These experiments were performed on the former occasion (vid. p. 21.) before

Dr. Hewett, Physician to St. George's Hospital,
Mr. Smyth, House Surg. and
Mr. Lane, Lecturer on Anatomy to that institution;
Mr. Oswald Beale,
Mr. Frederick Julius and
Messrs. Field, Veterinary Surgeons.

On the latter occasion, (vid. p. 30.) they were performed before

Mr. Babington, Surgeon to St. George's;
Dr. Burrow, Lecturer on Medical Jurisprudence to St. Bartholomew's;
Dr. Clark, Physician to the St. George's Infirmary;
Dr. Craghie of Edinburgh;
Dr. Elliotson, Physician to St. Thomas's;
Messrs. Field, Veterinary Surgeons;
Mr. H. J. Johnson, House Surgeon to St. George's;
Mr. F. Julius, Richmond;
Mr. Mayo, Surgeon to the Middlesex Hospital;
Dr. Latham, Physician to St. Bartholomew's;
Mr. Smyth, House Surg.

From experiments on small animals, supported by analogical arguments derived from pathology, I had previously been able to infer the nature of the heart's action, almost as I subsequently found it;* but the point required demonstration, and it appeared to me that the only possible mode of effecting this, was, by contriving to hear the sounds at the same moment that the actions were inspected and felt; since thus alone could it be unequivocally ascertained with what motions the sounds respectively coincided. Small animals I had found insufficient for the purpose; as, in them, the sounds are too indistinct, the motions too rapid, and the impulse too feeble, to afford satisfactory data. To large animals, therefore, I turned my attention, as presenting the only means likely to lead to a solution of the question.

As many may wish to follow this investigation through all its steps, and to form their own judgment from the data presented, I insert an abstract of the whole of the original experiments, as

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Dr. Watson, Professor of Forensic Medicine to King's College, and Phys. to the Middlesex Hospital; and

Dr. Williams, author of the "Rational Exposition of Auscultation," &c.

Mr. Brodie, who was accidentally absent, favoured me with an account of experiments by himself, which, so far as they went, coincided with my own. See his letter p. 37.

* Vid. p. 15, et seq.

published in the Medical Gazette, July 31, and August 21, 1830: and an account of the conclusions presented by a repetition of them on August 10th, 1831. The reader may, if he wish, omit these, and pass on to the next section.

Experiments, *July* 31, 1830.

As, in my opinion, it is impossible to avoid fallacies when the heart is pulsating at the rate of 200 per minute, I retarded the circulation of the rabbits which I examined by depriving them of sensibility previous to the operation. Each pulsation was thus resolved into several distinct and successive motions, which it is philosophical to regard as an analysis of the more rapid natural action. Under these circumstances I found the auricle to contract first—not slowly—but with a motion so *rapid* as to be almost instantaneous; the moment the fluid reached the ventricle, the latter was seen to start up, evidently by the contraction of its fibres on the fluid which it contained, and not by passive distention. This was more fully proved at a later period of the experiment, when the action of the heart was from time to time suspended, and the ventricle lay quiescent, though partially *distended with blood;* for, then, the auricle often made two or three contractions, which had no stimulant effect on the ventricle; while a fourth, not more violent than the preceding, and therefore not injecting more fluid, caused it to spring up in the manner already described. Simultaneously with the springing up, commenced the retraction of the apex towards the base, by which motion the apex was thrown forward, apparently in consequence of the long axis of the heart assuming a more horizontal position. These actions constituted only the commencement of the ventricular systole: its progress was marked by a further retraction of the apex and an approximation of the sides; while the whole ventricle was elevated further forward, and its long axis rendered still more horizontal, by the auricular distention, which advanced to its maximum in the same progression as the ventricle contracted to its extreme.

On examining the posterior aspect of the heart of a frog when its action was reduced to 15 or 20 per minute, the whole of the auricle, which had previously been concealed by the ventricle, being now exposed to view, it was found that, for a short space of time, the ventricle lay at rest *partially distended with blood;* the auricle then contracted with a smart brief motion—but only partially contracted, for the sinus venosus was constantly full of blood both in this experiment and those on the rabbit, and whether the circulation was quick or slow. When it had relaxed again, and not till then, the ventricle (stimulated, I conjecture, by the motion, for it certainly was not by distention) was seen suddenly to rise up on its basis, to shorten its fibres, and to expel its contents, which latter action was *slowly* performed. After the completion of the systole, which was indicated by the pale colour, the diastole took place, and allowed a partial influx of blood, denoted by the return of the red colour; and in this state the ventricle remained quiescent for a short space, until again stimulated by the auricular contraction. It may be objected to this account, that as the action of the heart was preternaturally slow, the motions were anormal. We thought, however, that we could discern the same series of actions when the pulsations were 40 per minute.*

Experiments, August 21, 1830.

I now proceed to the communication of further experiments, which, corroborated by pathological consideration hereafter to be adduced, will, I trust, be found decisive of the long controverted question respecting the cause of the motions and sounds of the heart.

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* These, and various other experiments detailed in the Lond. Med. Gaz. were performed at St. George's Hospital, in the presence of a number of the medical officers and other gentlemen attached to that institution. To Mr. Babington, Surgeon to St. George's, Mr. S. Lane, Lecturer on Anatomy to the Hospital, Mr. Smith, and Mr. H. J. Johnson, House-Surgeons, I am greatly indebted for their valuable aid in performing the experiments, and their patient and unbiassed scrutiny of the results.

At the conclusion of my experiments and researches hitherto detailed, I entertained the following impressions respecting the state of the question :—

That, in small animals, the auricular systole took place immediately *before* the ventricular, and not *after*, as supposed by Laennec, I regarded as certain, both from the evidence of my own experiments and from the concurrent testimony of the old physiologists. It was to be presumed that the same occurred in larger animals, but it remained to be proved.

That the *impulse* and *first sound* were referable to the ventricular, and not to the auricular contraction, I was equally persuaded, 1st, because the pulse, unquestionably a result of the ventricular systole, coincided so nearly, if not in every case perfectly, with the impulse and sound, that these three phenomena did not admit of being ascribed to any but the same cause; 2d, because clinical observations had proved to me, that certain anormal modifications of the heart's impulse and first sound corresponded with certain morbid conditions of the ventricular, but not of the auricular parietes.

That the *second sound* did not depend on the auricular systole, was indubitable; because the one was prior, and the other subsequent, to the ventricular contraction.

That it did not depend on the closure of the auriculo-ventricular valves was equally certain; because the closure of those valves takes place at the commencement of the ventricular contraction, whereas the sound occurs at its termination.

That it was not due to any other action of the auriculo-ventricular valves was obvious from physical considerations of their anatomical structure.

That it was not ascribable to the retrocession of the semilunar valves, I entertained a strong presumption, from having found the sound unimpaired, though the valves, on one side of the heart at least, were rigid with ossification; and the presumption amounted almost to certainty, from my having found the sound not only undiminished, but increased, in cases of enormous dilatation of both ventricles, in which it was impossible that the cavities could ever empty themselves; and where, consequently, the motion of the valves must have been impeded by the constant pressure of fluid on both sides (vid. for instance, case x.)

It remained, therefore, to be ascertained what was the cause of the second sound. As this sound is sometimes attended with *bruit de soufflet*, a phenomenon referable to the motion of the fluids, it was manifest that the natural sound, likewise, was in some way created by the same circumstance—the motion of the blood. Hence it became a question, what was the nature and cause of the motion of the blood accompanying the second sound: in more general terms, what was the state of the ventricles, with reference to the fluids, at the moment when the second sound occurred, and during the subsequent period of repose. This appeared to be the hinge of the whole question.

It was irrational to suppose that the ventricle, after its contraction, remained permanently braced during the interval of repose; as such a supposition would imply unintermitted labour of the muscle. I was therefore compelled to admit a relaxation, and this involved an ulterior admission, that of a diastole or expansion; not only because the relaxation of a muscle presupposes the elongation of its fibres, which renders a diastole inevitable, but because, granting the ventricles to be merely passive during their relaxation, the gravity of the blood in the auricles, aided by the vis-a-tergo, would cause its descent into the ventricles, and thus create a diastole. I had, furthermore, observed in the frog, when the heart was acting slowly, that the ventricular systole was followed by a diastole and partial distention with blood independent of the auricular contraction, and that the ventricle remained quiescent in this state of partial distention until again stimulated by the auricular contraction. These phenomena occurring during action of the heart rendered artificially slow, afforded a presumption (I do not say more) that the same occurred in animals the action of whose hearts was naturally slow.

Was, then, the influx of blood accompanying the ventricular diastole the cause of the second sound? An objection seemed to present itself in the circumstance that the sound was louder and smarter than could easily be accounted for on the supposition of its being produced by so indolent an action as the descent of blood merely by its own gravity and the venous vis-a-tergo.—An active dilating power appeared requisite. To suppose that this power resided in the muscular fibre, involved a contradiction, as it was tantamount to saying that a muscle could relax actively.* That it did not reside in the resiliency of the lungs was certain, because the heart continues to act though the lungs be collapsed. The only remaining cause to which an *active* dilating power could be referred, was, the ventricular elasticity, and this appeared scarcely adequate to the production of the effect.

Notwithstanding these apparent objections, pathological observations, corroborated by what I had seen in the frog, led me to infer that an influx of blood into the ventricles at their diastole *did* occur, and with sufficient energy to produce the sound in question;—for I had found that, in hypertrophy and dilatation, the second sound was accompanied by a very strong shock, which, from its appearing to be of a *receding* nature, I have, for five or six years, designated in my case-books by the epithet *back-stroke;* and this motion, and the accompanying sound were, *cæteris paribus*, stronger in proportion as the hypertrophy and dilatation were greater.* Still, however, that the influx of blood during the ventricular dilatation, was the cause of the second sound, was only conjectural, and it required demonstrative proof before it could be assumed as a fact.

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* I believe, however, that this point is open for further investigation.

How, then, was this, and the collateral questions, to be determined?

It appeared to me that the only possible mode was, by contriving to hear the sound at the same moment that the action of the heart was inspected and felt; since it could thus be unequivocally ascertained with what motions the sounds respectively coincided. Small animals were obviously insufficient for this purpose, as, in them, the sounds are too indistinct, the motions too rapid, and the impulse too feeble, to afford satisfactory data. To the larger animals, therefore, I at once turned my attention, as presenting the only means likely to lead to a solution of the difficulty.

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* The auricular impulse was not overlooked by Laennec, but I am not aware that he, or any other writer, has noticed the *back-stroke* as a sign of disease.—*Vide Traité de l'Auscult. 2de édit. tom.* ii. *p.* 395.

The whole subject, then, seemed to resolve itself into the following questions, which I drew out and proposed to my coadjutors, before the operation, as the points for investigation:—

1. Do the auricles contract immediately before the ventricles?
2. Does an interval occur between the two contractions, or is the succession so rapid as to amount to continuity of action?
3. Does the ventricular contraction cause the impulse, pulse, and first sound?
4. Do the ventricles contract completely, and do they remain closed and empty, during the interval of repose? Or—
5. Do the ventricles dilate again immediately after their systole; and is this dilatation attended with an influx of blood from the auricles?
6. Is the influx of blood into the ventricles during their diastole the cause of the second sound? If not—
7. What is the cause of the second sound?

Experiment I.—An ass, of which the pulse and impulse were 48 per minute, was instantaneously deprived of sensation and motion, by a smart blow on the head. The trachœa was opened, a large bellows-pipe introduced, and artificial respiration maintained; while, at the same time, the left ribs were sawn through near the sternum, and forcibly bent back and broken,* so as widely and completely to expose the heart immediately behind the left shoulder: the whole was accomplished in less than five minutes.

The pericardium was next opened, and the auricles and ventricles fully displayed. The action of the heart was at first quick, tumultuous, quivering, and irregular; but after the lapse of about three or four minutes, it became regular and slower. The auricle was now seen to contract *first*, and the ventricle instantly afterwards; or, in more descriptive language, a slight contractile motion, accompanied with very inconsiderable diminution of volume, was observed to commence in the auricle, and to be propagated rapidly to the ventricle. It was not, however, so quick that it could not easily be followed by the eye; yet it seemed to be rather a continuity of action, than to consist of two consecutive parts.

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* This plan was adopted in preference to cutting, in order to obviate hæmorrhage from the intercostal vessels.

The ventricular contraction appeared, and was felt by the hand, to consist of a sudden energetic jerk, accompanied with a depression of the centre or body of the ventricle. This contraction was heard (through the stethoscope, applied *immediately* to the organ) to be accompanied by the ventricular sound. A note was accordingly dictated,* that, 1. *The ventricular sound was heard whilst the ventricle was seen to contract.* At an interval of time equal to that which intervenes between the first and second sounds of the heart, the contraction was followed by a sudden, jerking re-expansion or diastole, which appeared *to elevate the body of the ventricle more than the previous contraction.* Hence one of the party (Mr. Lane) expressed his opinion that it was the *diastole*, and not the systole, that occasioned the impulse. This opinion rendered it necessary instantly to repeat all our observations. The stethoscope was accordingly resumed, and several times applied by Mr. Field and the writer alternately, each counting 1, 2, synchronously with the sounds which he heard and the impulse communicated to his ear; while others applied their hands to the ventricle, and at the same time inspected its motion. It was now proved, to the perfect satisfaction of Mr. Lane and all present, that the sound 1, and the impulse felt by the auscultator, coincided with the *visible* depression (*i. e.* contraction) of the ventricle, and the impulse felt by the hand. It was therefore dictated that, 2. *When the action of the heart was become slower (supposed to be about forty per minute) and was becoming feeble, the ventricular systolic sound and the impulse were heard, seen, and felt, both by the ear and hand, to be simultaneous.*

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* The notes were written by Mr. F. Julius to the conjoint dictation of the party, during the progress, and immediately after the conclusion of each experiment; and they were finally revised and signed.

At an early part of the experiment it had been unanimously agreed that the ventricle never contracted fully, though it was then acting with great power. It was, therefore, dictated that, 3. *The ventricle never contracted fully.*

4. *It remained apparently full during the intervals of repose.* (*i. e.* from the conclusion of the diastole to the commencement of the ventricular contraction).

On interposing the hand between the apex of the heart and the rib, which had been left above that part, the fingers were struck vigorously by the apex at the moment that the body of the ventricle was in the act of retraction.

As the action of the heart, after ceasing to be tumultuous, became somewhat feeble, the second sound was never very audible. It was distinctly heard, however, by Mr. Field and the writer; but as the others could not satisfactorily recognize it, a general note was deemed inadmissible, and a by-note only was dictated, the point being reserved, for further investigation, at the next experiment.

By-note.—*Mr. Field and Dr. Hope listened with the stethoscope alternately, and counted* 1, 2, *in unison with the sounds which they heard; while the others saw that* 1 *coincided with the ventricular systole, and* 2 *with its diastole.*

This first experiment was not considered conclusive. In consequence of the turbulence of the heart's action at first, and its feebleness at last, the time favourable for observation was too brief; and, consequently, a majority of the party had not complete confidence in the accuracy of their observations. This diffidence was shown by the second experiment to be greater than the case warranted.

The second experiment was performed immediately after the first.

Experiment II.—The heart of an ass was exposed to view in the same manner as before, but with still greater celerity. For *about a minute* *only* the action was quivering and irregular; it then fell to its natural standard (40 to 50 per minute), became *perfectly regular*, and the ventricular contraction, as felt by the hand and the stethoscope, was performed with a power which can scarcely be imagined from an examination on the outside of the chest.

Three successive motions—namely, the auricular systole, the ventricular systole, and the ventricular diastole—were now distinctly recognized and acknowledged by all who witnessed them. The stethoscope was applied to the ventricle, and the *two* sounds were clearly and unequivocally heard even by those who were unaccustomed to the instrument. Five gentlemen listened deliberately twice over, and two of them, three times, before it was dictated that, 1st, *Drs. Hewett and Hope, and Messrs. Lane, Field, and Cooper, listened successively through the stethoscope applied to the ventricle, and severally counted* 1, 2, *synchronously with the sounds which they heard; while the others ascertained, by the touch and sight, that the sound* 1 *coincided with the ventricular systole, and the sound* 2 *with its diastole.*

This part of the experiment was so deliberately performed that it occupied from ten minutes to a quarter of an hour, as near as could be judged from the whole time expended (from twenty to twenty-five minutes), and each of the experimenters was asked whether he was satisfied, whilst he had still an opportunity of renewing his examination.

It was now submitted to investigation, how the ventricular systole could occasion the impulse; since the body of the organ appeared to *recede* during that motion. The result was the following note:

2. *While the ear rested on the stethoscope applied to the middle of the ventricle, the impulse was felt by the auscultator to coincide with the systole, notwithstanding that the body of the ventricle appeared to be receding at the moment the impulse took place.*

During the course of the experiment the action of the auricle was again examined. Its anterior edge and surface only were in sight, the root and sinus being concealed behind the ventricle. It was noted that—

3. *The auricle never emptied itself, and its contraction was always very inconsiderable. The anterior edge and surface were seen to retract with a rather sudden motion; but as the extent of the motion was very inconsiderable, it had the appearance of being feeble.*

The contraction of the auricle was so much less than there was reason to anticipate from the extent of its action in smaller animals, that it was questioned whether it was, in the present instance, performed with the natural vigour. The extraordinary power with which the ventricle acted, favoured the affirmative; and as the proportion of the auricle to the ventricle is singularly less in large animals than in small, there is reason to suspect that they perform a less important function in the former.

The inevitable conclusions deducible from these experiments are, that—

Of the Motions of the Heart—

1. The auricles contract so immediately before the ventricles, that the one motion is propagated into the other, almost as if by continuity of action; yet the motion is not so quick that it cannot readily be traced with the eye.
2. The extent of the auricular contraction is very inconsiderable, probably not amounting to one-third of its volume. Hence the quantity of blood expelled by it into the ventricle, is much less than its capacity would indicate.
3. The ventricular contraction is the cause of the impulse against the side; first, because the auricular contraction is too inconsiderable to be capable of producing it; second, because the impulse occurs after the auricular

contraction, and simultaneously with the ventricular, as ascertained by the sight and touch; third, because the impulse coincides with the pulse so accurately as not to admit of being ascribed to any but the same cause.

4. It is the apex of the heart which strikes the ribs.

5. The ventricular contraction commences suddenly, but it is prolonged until an instant before the second sound, which instant is occupied by the ventricular diastole.

6. The ventricles do not appear ever to empty themselves completely.

7. The systole is followed by a diastole, which is an instantaneous motion, accompanied with an influx of blood from the auricles, by which the ventricles re-expand, but the apex collapses and retires from the side.

8. After the diastole, the ventricles remain quiescent, and in a state of apparently natural fulness, until again stimulated by the succeeding auricular contraction.

Of the Sounds.

9. The *first sound* is caused by the systole of the ventricles.

10. The *second sound* is occasioned by the diastole of the ventricles.

Of the Rhythm.

Order of succession—

1. The auricular systole.
2. The ventricular systole, the impulse, and the pulse.
3. The ventricular diastole.
4. The interval of ventricular repose, towards the termination of which the auricular systole takes place.

Duration.

This is the same as indicated by Laennec, viz.

The ventricular systole occupies half the time, or thereabout, of a whole beat.

The ventricular diastole occupies one-fourth, or at most one-third.

The interval of repose occupies one-fourth, or rather less.

The auricular systole occupies a portion of the interval of repose.

Experiments repeated, August 10, 1831.

Three asses were successively made the subject of operation, the process being conducted as before. It may be premised that, in consequence of the percussion of the brain not having been, in the first instance, sufficiently smart, the action of the heart was, in all three cases, more or less irregular through the greater part of the experiment, not continuing, as on the former occasions, ten or fifteen minutes almost without the slightest intermission. Notwithstanding, as the action was maintained for an equal, if not longer time, the periods of regular pulsation were sufficiently numerous and prolonged to allow of deliberate examination. The irregularity led to one important discovery which had hitherto escaped me: namely, the movements of the ventricles with their corresponding sounds continued perfect while the auricles were motionless.

The following queries were circulated to the individuals present a few days previous to the experiments. They were severally read after each of the three experiments, and the answers were the conjoint dictation of the party, partly during the experiments, and partly at the successive recapitulations.

1. *Do the ventricular systole, the first sound, the impulse, and the pulse coincide?*

A. They coincide perfectly, except that sometimes there appeared to be a *barely* appreciable interval between the impulse or first motion of the ventricle (as seen, and also felt with the fingers interposed between the apex and the ribs) and the pulse in the radial artery under the shoulder.

Remark.—The interval alluded to was ascribed to the distance of the artery from the heart. In more remote arteries it is proportionably greater, and in those near the heart it does not exist at all.

2. *Do the ventricles expel the whole, or a part only, of their contents; and what is their state during the interval of repose? Are they full or empty?*

A. The ventricles not being transparent, it is not demonstrable whether they expel the whole of their contents; but the diminution of their volume by the systole is not in general so great as to convey that impression. During the interval of repose they are full, being restored to that state by the diastole. By *fulness*, is not meant *distention*, this being an ulterior degree.

Remark.—The question whether the ventricles expelled the whole of their contents or not, originated in an opinion, which had been maintained, that they did so, and, by the collision of their internal surfaces, occasioned the second sound. As this sound is proved to result from the diastole, the question becomes redundant, and its determination unimportant.

3. *With what motion of what part does the se-*

cond sound coincide, and what is its cause? Is it the ventricular diastole?

A. The second sound coincided with a motion, sensible to the touch and sight, by which the ventricle returned from its systole to the same state, with respect to size, form, and position, as before the systole. This motion was the relaxation or diastole.

4. *Do the auricles contract before, or after the ventricles, with respect to the interval of repose?*

A. Evidently before, being instantly followed by the ventricular systole. The interval of repose distinctly falls between the ventricular diastole and the auricular systole, the repose of the ventricles continuing through the auricular systole to the next ventricular systole. Such were the phenomena observed during the short periods when the motions of the auricles were regular; but, for the most part, there was either no perceptible motion in them, or the motions were irregular and bore no relation whatever to the ventricular movements.

Remark.—From subsequent experiments on rabbits, in the performance of which I was favoured with the assistance of Dr. Hewett, and Mr. Daniel, Surgeon, of Ramsgate, I am led to believe that the irregularity of the heart's action is an incidental circumstance, dependent on the mode in which the animal is stupified, and artificial respiration maintained: consequently, that it is capable of being obviated. At the suggestion of Mr. Brodie, I stupified the rabbits by inoculating them with woorara poison. In the first experiment, after the expiration of a few minutes stupefaction took place so suddenly that the action of the heart was irrecoverably extinct before artificial respiration could be established. In a second instance, arrangements having been made to establish it more expeditiously, the action of the heart was maintained in the greatest perfection, after the cerebral life of the animal had become completely extinct. We now found that, on temporarily suspending the respiration, the heart instantly became gorged, of a black colour, and distended to nearly double its natural size, while its motions were either an irregular, occasional flutter, or were wholly suspended. On resuming the inflation, the motions gradually became more and more frequent, extensive, and regular, while the distention and blackness decreased in the same proportion; until, at length, the organ regained its previous colour and dimensions, and beat with its accustomed energy and regularity at the rate of 200 per minute. This process was repeated again and again for nearly an hour; and more than once, the action was renovated, though with difficulty, after both the ventricles and auricles had rested some seconds in a state of complete immobility. Hence it appears that, when the stupefaction is complete, as it is from woorara poison, and artificial respiration is adequately maintained, the action of the heart may be kept regular: and it was from greater success in these two circumstances that, in my first experiments on asses, the regularity was so remarkable. The hammer employed had a smaller head, its surface, which was slightly excavated, not exceeding an inch in diameter. By this, a corresponding portion of the skull was depressed, whence the extinction of cerebral life was instantaneous and complete, and thus the performance of artificial respiration was rendered more easy. I mention these particulars, in order that, should it be found necessary to repeat the experiments, an unnecessary destruction of life may be avoided. I may add, that the experiments on the rabbit afforded an instructive exemplification of the manner in which congestion of the heart takes place in paroxysms of excessive dyspnœa and in the agony of death. They also showed how, under these circumstances, both the impulse and sounds, even of the most enlarged heart, may be diminished or become totally extinct. To return from this digression—

5. *Do the auricles contract slightly or extensively?*

A. Very slightly, and principally at its appendix, the motion running vermicularly into the ventricular systole.

Remark.—When several irregular ventricular contractions followed each other rapidly, the corresponding diastoles were attended with a slight retraction of the auricles, most conspicuous at their sinuses. This phenomenon proceeded from the increased suction of blood by the ventricles.

6. *Are the auricles ever empty, or are they constantly full?*

A. Constantly full, their motions ranging between fulness and distention.

The following dictations formed a corollary.

"The first and second sounds were heard, and the corresponding motions (the systolic and diastolic) were felt, while the auricles were not contracting."

Remark.—Had this observation been made in my first experiments, it would have superseded the necessity for much reasoning, as it conclusively fixes the sounds, the impulse, and the *back-stroke*, on the ventricles.

"When the heart was gorged, towards the conclusion of the experiments, the first sound only was heard."

Remark.—At the same time the action was very feeble. This, as in the experiment on the rabbit, displays the cause of the diminution of sound and impulse in suffocative dyspnœa and on the supervention of death.

Mr. Brodie, finding himself unable to attend the experiments on the 10th, favoured me with the following communication on the evening of the 9th. If doubt remains on the mind of any respecting the points in my experiments to which his observations refer, they cannot fail to have the weight which attaches to any thing that proceeds from the pen of this distinguished physiologist.

My dear Sir,

—— With respect to some of your propositions, I think that I can already solve them in a way satisfactory at least to myself. 1. When I was making experiments on the circulation formerly, it appeared to me that the pulse and the systole of the ventricle exactly coincided. 2. It appeared to me that, when the action of the heart was vigorous, the ventricles emptied themselves at each contraction; but that they did not do so, when the action of the heart was feeble. 3. I never found the auricles completely empty themselves, nor did I, in dogs, rabbits, &c. ever observe in them any regular systole corresponding to, and alternating with, that of the ventricles. I often used to observe several slight contractions of the auricle, especially of the appendix of the auricle, for one of the ventricle. In frogs, however, I have a strong recollection that the actions did alternate and correspond, but, not being able to find my notes, I cannot speak positively.

If I were to institute such a series of experiments, I would first stupify the animal by inoculating him with the woorara, or some poison of the same kind. You will observe that when an animal is stupified with the woorara, there is no struggling, and you may maintain the heart's action, by inflating the lungs, for an indefinite period. I have some woorara, and can, I doubt not, furnish you with enough for the experiment.

I am, dear Sir,
Yours very truly,
B. C. Brodie.

The woorara arrived too late for the experiments on the asses. I have already described how well it subsequently answered on a rabbit. Prussic acid was tried on one ass, but the animal recovered from four or five successive drachms given by the mouth.

According to the data supplied by the above experiments and by the cases appended to this work, the physiological and pathological phenomena of the heart's action appear to be as follows.

SECTION II.

PHYSIOLOGICAL PHENOMENA OF THE HEART'S ACTION.

I. *The Phenomena in the Order of their occurrence; viz. The Auricular Systole*, 39; *Ventricular Diastole; Interval of Repose of the Ventricles, and of the Auricles*, 40; *Rhythm*, 41.

II. *The Causes, Mechanism and Objects of the Motions. Haller unable to explain them*, 41. *Auricular Systole*, 41; *Ventricular*, 42. *Proportion of Blood expelled*, 42. *Mechanism of the Impulse*, 42. *Diastole*, 44. *Interval of Repose*, 45; *Length of that of the Ventricles*, 45; *and of the Auricles*, 46.

III. *The Causes and Mechanism of the Sounds*, 47. *Muscular Sound not the Cause*, 47. *Motions of the Fluids, the Cause*, 48. *Explanation, of the first Sound*, 48; *of the Second*, 49. *Auricles do not produce Sound*, 49.

I. *The phenomena of the heart's action in the order of their occurrence.*—The first motion of the heart which interrupts the interval of repose, is the auricular systole. It is a very slight and brief contractile movement, more considerable in the auricular appendix than elsewhere, and propagated, with a rapid vermicular motion, towards the ventricle, in the systole of which it terminates rather by continuity of action, than by two successive movements.

The ventricular systole commences suddenly, and terminates in the diastole, which is marked by the second sound. Synchronous with the systole are, the first sound, the impulse of the apex against the ribs, and, in vessels near the heart, the pulse; but, in vessels at some distance, as the radial, the pulse follows at a barely appreciable interval.

The systole of the ventricles is followed by their diastole, during which they return, by an instantaneous expansive movement sensible to the touch and sight, to the same state (with respect to size, shape, position &c.) as during the previous interval of repose. This movement or diastole is accompanied by the second sound, by an influx of blood from the auricles, by a slight retractile motion of these cavities most observable at their sinuses, and by a retrocession of the apex of the heart from the walls of the chest.

Next succeeds the interval of repose, during which the ventricle remains at rest, in a state of fulness, though not distention, through the whole period intervening between the second and the first sounds; but the auricle remains at rest during the first portion only of that period, the remainder being occupied by its next contraction, with which recommences the series of actions described.

The rhythm of the heart, that is, the duration of the several parts of this series, which constitute what may be called a beat, is the same as described by Laennec: viz, 1. The ventricular systole occupies half the time, or thereabouts, of a whole beat. 2. The ventricular diastole occupies a fourth, or at most a third. 3. The interval of ventricular repose occupies a fourth, or rather less, during the latter half of which the auricular systole takes place.

II. *Causes, mechanism, and objects of the motions.* Though Haller accurately noticed the motions of the heart, he was unable to account for the particular order of their occurrence. Hence he says "The reason is a postulatum (postulatur ratio) why, first, the right and simultaneously the left auricle contract, while in the meantime the ventricles rest relaxed; why, a little after, the auricles are relaxed, but the ventricles contract; and then, in a third portion of time, the ventricles *repose* relaxed, but the auricles again smartly contract."* The reasons required can now be assigned. The auricles, which are always in a state of fulness though not distension, arrive, from the progressive influx of blood during the first portion of the ventricular repose, at the state of distention, by which they are stimulated to contract. The object for the contraction at this moment, is, to propel a small additional quantity of blood into the ventricles, already full, for the purpose of bringing them from the state of mere fulness to that of distention:—an object which could not be accomplished without a contraction, as the blood could not otherwise force its way into the ventricles against the resistance offered by their elastic parietes. These cavities, then, being brought to the state of distention, are thereby stimulated to contract. They expel a greater or less proportion of their contents—the whole in small animals, frogs for instance,—as is proved by the ventricles becoming pale; but in large animals, as the ass, they do not *appear*, judging from the diminution of size, to expel the whole, though, as the ventricular walls are opake, whether they do or not, does not admit of demonstration. During the act of expulsion the apex is tilted forwards and upwards and occasions the impulse against the ribs. On the mechanism of this motion, a point hitherto much disputed, it is necessary somewhat to enlarge.

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* De Motu Cordis. Lugduni Batavorum. 1737. p. 37.

When the heart of an animal, as a frog, rabbit, dog, turtle &c. is detached from the body before organic life is extinct, and placed upon a table, it continues to act, and each contraction elevates the apex. Hence it is unquestionable that the muscular fibres have an inherent faculty of producing this action. The manner in which the action is accomplished is very visible on inspection. During the state of relaxation, the heart lies collapsed and flattened, with a large extent of its under surface applied to the table; on contracting it starts up, and, assuming a more rounded form, is sustained by a comparatively small point of contact. The apex is, consequently, elevated, and the elevation is greater in consequence of the base, from its superior weight, being the more fixed part. The action is closely analogous in the living subject. Before describing it, I shall advert for a moment to the anatomical disposition of the parts of the heart.

In large animals, as the human species, the auricles, especially the left, are attached to the posterior part of the base, and the aorta and pulmonary artery spring from its anterior part. These vessels are the fixed points towards which the fibres of the heart contract during the ventricular systole, and their stability is increased by the injection and distention which they undergo during the systole. The sinuses of the auricles being constantly full, even during the contraction of the appendices, and regurgitation of their contents into the veins being opposed by the elasticity of the venous coats, by the pressure of the surrounding parts, by the capillary vis-a-tergo, and by the atmospheric pressure, with a power exceeding the weight of the ventricles, the auricles form an almost unyielding fulcrum beneath the ventricles during the systole of the latter.

Such being the anatomical and physiological state of the parts, during the ventricular systole the braced fibres, contracting towards the aorta and pulmonary artery in front, draw the tense and rounded body of the ventricles upon the auricular sinuses behind. Consequently, the apex of the ventricles is tilted up; and this motion is performed with considerable velocity, because, if I may be allowed the illustration, the apex is the long arm of the lever, the auricles being the fulcrum, and the moving power at the aorta and pulmonary artery. In proportion as the ventricles contract to their extreme, the apex is thrown more and more forward by the auricular distention, advancing in the same progression to its extreme. Another circumstance probably contributes to the elevation of the apex; namely, the retropulsion of the auricular valves: for, as these act on a column of fluid which offers a resistance greater than the weight of the heart, the action is reflected on the organ itself, and impels it forward.

The diastole appears to be occasioned by several concurrent causes; viz. 1. That power of the muscle (whether elasticity or something more, is unimportant) by which it reverts from the state of contraction to that of relaxation, and in virtue of which it exercises a degree of suction. 2. The distention of the auricles, which is greater at the moment of the diastole than at any other, as they have been filling during a longer period—namely, that of the ventricular contraction or half of a whole beat. 3. The weight of the ventricles collapsing from their systole on the distended auricles beneath them. 4. The width of the auriculo-ventricular orifice, which allows the blood to shoot in with instantaneous velocity. It is manifest that as so many powerful causes conspire to effect the influx of the blood, an auricular contraction at this time, and for this purpose, as imagined by Laennec, would be superfluous. The draught of blood from the auricles during the diastole, causes the slight retraction of these cavities observable at that moment.

The object of the interval of repose, is, to afford rest to the organ, and no arrangements could answer this object so completely as those described. We see that the ventricles, by their diastole, are brought to the state most favourable to their repose—that of natural fulness without distention, and in this state they remain, to employ an approximative calculation very nearly exact, one fourth of each beat or six hours in twenty-four; but, if we admit that the diastole also is a kind of repose to the ventricles, we must add another fourth, making the whole period half a beat or twelve hours, which is the period assigned by Laennec.[*] Had the auricular systole succeeded the ventricular and immediately brought the ventricles from the state of contraction to that of distention, there could have been no repose, as distention implies the exertion of the tonic power of a muscle, which, as is well known to those who have to reduce dislocated joints, is eminently productive of fatigue. Nor, indeed, could any *interval* of action then have taken place; for, admitting distention to be the stimulus of the ventricles, their contraction would have ensued instantly on their being brought into the state of distention. We see, further, according to the theory which I advocate, that the auricles, by evacuating their contents into the ventricles at twice instead of once, avoid the excessive distention at which they must have arrived had the blood been accumulating in them during three fourths of a beat. Although their systole occupies only about one eighth of a beat, or half the time between the second and first sounds, the whole of the remaining seven eights is not devoted to repose; for, during the greater part of this time, the auricles are in a state of greater or less distention, which, as above stated, is not repose. Under these circumstances it is too hypothetical, if not impossible, to estimate by numerical calculation the exact amount of the auricular repose, but analogy, countenanced perhaps by a rude calculation, leads us to infer that it must be about equal to the ventricular. Laennec estimates it at one half more, or eighteen hours in twenty-four, but this he does on the assumption that the auricular contraction occupies one fourth of a beat, and without allowing for the fatigue occasioned by the state of distention into which the auricles are brought during a portion of the intervals between their contractions.

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[*] De l'Auscult. tom. ii. p. 408.

III. *The causes and mechanism of the sounds.*—Laennec does not offer any explanation of the mode in which the natural sounds of the heart are generated. A morbid sound, the bellows-murmur, he contends may be produced by the movement of the muscular fibres of the organ when they contract *spasmodically*—a subject which will be considered in the next section; but, admitting for the sake of argument that his observation is true, such a cause would be totally insufficient to account for the natural sounds, as these are not only different in their character, but, beyond comparison, louder than the loudest "*muscular sounds*" that can be produced by any exertion of the most powerful muscles in the body,—a disparity the more striking, as the sounds of the heart are generally louder in direct proportion as the ventricular walls are thinner. Moreover, this cause would not account for the second sound; as it takes place during the act, not of contraction, but of relaxation. This argument of course assumes that the diastole is not caused by an *active* muscular agency.

All the phenomena of the heart's action, both in health and disease, lead me to believe that the sounds are occasioned by the motions of the contained fluid, and the mechanism of their production I conceive to be, according to the laws of physics, as follows. When the ventricles contract, an impulse is given to the particles of fluid in contact with them; and this being propagated by collision from particle to particle, generates sound. The irregularity of the interior surface, occasioned by the columnæ carneæ, is calculated to favour this formation of sound; for, on the very first contractile movement, the stratum of fluid next to the surface, and involved in the sinuosities of the columnæ carneæ, is thrown into an infinity of conflicting currents; whence the collision of particles is more extensive and violent, than if it were occasioned merely by a simple direct impulse. It will be understood that these conflicting currents do not neutralize each other by reciprocal reaction, because, as the blood finds egress by the arterial orifice, the reaction is not equal. If the contraction be gradual, the sound will, for obvious reasons, be subdued and prolonged, and if it be sudden, the sound will be proportionably short, loud, and clear. While this is going on at the surface of the fluid, the central mass is tending towards the aortic and pulmonary apertures; and, as its current is compounded of a multitude of conflicting streams, reflected on every side from the ventricular walls and converging towards the orifices, the vibratory collision thus occasioned amongst the particles of blood, produces sound.

The second sound or that of the ventricular diastole, is generated by a more simple mechanism, and is, consequently, more uniform in its character. When the diastole takes place, the blood, put in motion by a number of concurrent circumstances already particularised, shoots with instantaneous velocity from the auricles into the ventricles; and the reaction of the ventricular walls on its particles, when their course is abruptly arrested by the completion of the diastole, is, I conceive, the cause of the loud, brief, and clear sound.

The auricles do not contribute to the production of either of the sounds; as, in the experiments alluded to on the ass, they were heard in equal perfection when the auricles were in a state of immobility. Nor does the auricular contraction, in my opinion, produce any sound whatever; for the movement, in large animals at least, is too inconsiderable to be capable of it, and no third sound of the heart is ever heard.

SECTION III.

PATHOLOGICAL PHENOMENA OF THE HEART'S ACTION.

I. *Modifications produced in the Motions and Sounds, by Hypertrophy,* 50—*by Dilatation,* 52—*by Hypertrophy with Dilatation,* 53—*by valvular Disease,* 54. *Circumstances under which Sounds and Impulse may be diminished in any Disease of the Heart,* 55.

By hypertrophy, the impulse is increased and the sounds diminished. "The impulse," says Laennec, "is ordinarily sufficiently strong to heave the head of the observer in a very sensible manner, and sometimes it is so strong as to produce a shock disagreeable to the ear. The greater the hypertrophy, *the more time that heaving takes for its performance,* and, when the disease is carried to a high degree, we evidently perceive that it takes place by a *gradual progression;* it seems as if the heart swelled and applied itself to the walls of the chest, at first by a single point, then by its whole surface, and, in the next place, suddenly sunk back" (s'affaisse.)[*]

Of the sounds he says "In moderate hypertrophy the contraction of the ventricles produces only a stifled sound analogous to the murmur of inspiration, and the clack *(claquement)* of the auricles is much less sonorous than in the natural state. In hypertrophy carried to an extreme degree, the contraction of the ventricles produces nothing but a shock without sound, and the sound of the auricle, become extremely dull, is scarcely audible."[†]

The causes of these modifications are very intelligible. The power of the impulse is increased in the direct ratio of the hypertrophy; and the movement is a progressive heaving, because the hypertrophous ventricle contracts slowly and with a gradual progression. For the same reason the first sound is diminished,—the impulse communicated to the contained fluid not being sufficiently smart to occasion more than a dull, stifled sound, if any at all. The second sound is dull and weak because the unyielding thickness of the ventricular walls renders their expansion by the fluid more difficult and gradual;—consequently, the reaction, generating the sound, is more languid. When the hypertrophy is extreme and the cavity contracted, the sound becoming extinct, not only from the operation of the same causes in a greater degree, but also from the less copious influx of blood.

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[*] De l'Auscult. edit. 2. tom. ii. p. 395.

[†] Ibid. p. 400.

By dilatation, the impulse is diminished, often to the extent of being imperceptible. When perceptible, it is a sudden brief blow, which communicates a shock or vibration to the thoracic walls, but has not power or duration to elevate them. The reason is, that, as a thin muscle has less power, but greater facility and rapidity of motion, than a thick one, the attenuated ventricles contract on their contents with greater velocity than natural, but their action is more feeble: accordingly, the impulse is diminished, and its power is sooner exhausted,—whence the brevity of the shock. The apex, in other words, is suddenly tilted forwards, and as suddenly collapses.

The first sound in dilatation, becomes loud, brief, and clear, like the second. This arises from the impulse communicated to the blood being smart, though feeble;—a species of impulse which, by causing rapid, but fine vibrations, produces a sound of that description, whether liquids or air be the vibrating medium. Thus,—to employ a familiar illustration, instead of referring to abstract laws of physics,—let the reader clap the hollow palms transversely together, so as to include a portion of air, and thus produce a flapping sound. If the motion be performed smartly, though with little muscular power, the sound will be loud and clear: if it be performed slowly, whatever be the degree of muscular power exerted, the sound will be subdued and on a lower key. An additional cause for the loudness of the sound in dilatation, probably is, that the tenuity of the walls facilitates its transmission from the interior of the ventricles. The first sound in dilatation is not prolonged, as in the healthy heart, by the currents of fluid converging to the ventricular orifices, because the currents are too sluggish to occasion sound. One proof of this sluggishness consists in the feebleness, softness, and sometimes smallness of the pulse.

The second sound also, is increased; because the quantity of blood entering the ventricles during their diastole is probably augmented, and, from the thinness of their walls, the check is more sudden.

By hypertrophy with dilatation.—The modifications occasioned by this affection are compounds of those of hypertrophy and those of dilatation. The contractions of the ventricles give a strong impulse—"abrupt, dead, (sec,) violent blows, which strongly repel the hand:"* they partake, in short, of the power of hypertrophy and the smartness of dilatation. The first sound is increased, and audible at a great distance; for it consists of the loud, abrupt sound of dilatation, blended with the murmur resulting from the powerful propulsion of the blood occasioned by hypertrophy. The second sound also is louder than natural, because an augmented quantity of blood enters during the ventricular diastole, and because its momentum is increased by the preternatural weight with which the heart collapses on the auricles, and probably, also, by an augmentation of diastolic power in the ventricles themselves.

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* Laennec de l'Auscult. tom. ii. p. 515.

Hypertrophy with dilatation is not unfrequently accompanied with bellows-murmur, an exposition of the cause of which I reserve for the article on murmurs.

By valvular disease the sounds acquire various morbid murmurs, as those of bellows, of filing, rasping, sawing, whistling, &c., which it is unnecessary here to describe, as they are treated of in the article on Diseases of the Valves. I shall merely recapitulate the circumstances under which they occur for the purpose of showing that they assimilate with, and substantiate, the view which I have taken of the heart's action.

When the *aortic, or the pulmonic valves* are contracted, a morbid murmur accompanies the sound of the ventricular systole: and when the valves, not closing accurately, admit of regurgitation, a murmur accompanies the sound of the ventricular diastole also; but in the latter case it is extremely slight and brief, because, as I imagine, the swift influx of blood from the auricle during the diastole, almost instantly puts an end to any regurgitation capable of producing sound. When the *Mitral* or the *Tricuspid* valve is contracted, a murmur accompanies, and sometimes entirely supersedes, the second sound, being occasioned by the obstructed passage of the blood from the auricle into the ventricle during the diastole of the latter. When the valve, not closing accurately, admits of regurgitation, a murmur accompanies the first sound. This fact was one of the very few overlooked by that wonderfully accurate observer Laennec. It was noticed by the writer in 1825, and the number of cases with which he is enabled to substantiate it, leads him to assume it as certain. Vid. cases.—Chr. Anderson, Dennis.

The impulse and sounds, in any affection of the heart, may partially, or even totally fail, when the organ, either from its own debility, or an obstacle in the course of the circulation, is gorged with an accumulation of blood which exceeds its propulsive power; and also when the vital powers are reduced by any cause whatever. This is not only indicated by pathology, but is demonstrable on the stupified living animal; for, if artificial respiration be temporarily suspended, the diminution of sound and impulse immediately takes place, and it may thus be produced and removed at pleasure. The heart, during the intervals of inflation, is seen in a gorged state, scarcely either contracting or dilating. See *Experiments*, p. 34.

II. *The Mechanism of morbid valvular Murmurs*, 56. *Exemplified in Tubes*, 56. *Murmur from Ossification*, 57. *Circumstances under which Murmur is the loudest*, 57. *How occasioned by an infundibuliform Auriculo-ventricular Valve*, 58. *By a tense Valve with a central Perforation*, 59. *By Regurgitation through a projecting Valve*, 59. *Illustrations from Hydraulics*, 59. *Why Contraction of an Auriculo-ventricular Valve may occasion Murmur of the first, and not of the second Sound*, 61. *Slight Contraction does not produce Murmur of the second Sound*, 62.

II. *The mechanism of morbid valvular murmurs.*—Valvular murmurs are occasioned by collision of the particles of the blood, when this fluid is, by any cause, thrown into preternatural commotion during its passage through the orifice of a cavity. To offer an experimental exemplification of this—a similar murmur is produced when water is transmitted with sufficient velocity through a tube, in any part of which there exists an internal prominence or contraction of its calibre. The same occurs when the leather pipe of a fire-engine is slightly compressed with a finger; or, when similar compression is exercised with the stethoscope on a superficial artery of primary or secondary magnitude.

Obstructions of a hard, rugged kind, as ossifications, occasion louder murmurs than smooth obstructions, because they more completely break the current of the blood. Murmurs are not, as is often supposed, louder, cæteris paribus, in proportion as the valvular contraction is greater. On the contrary, the loudest murmurs are produced by a moderate contraction, and they become weak when it is extreme. Thus, a rugged osseous concretion, the size of an ordinary pea, in the aortic orifice, I have found to produce the loudest possible murmur; whereas, a contraction of the mitral or tricuspid valve to the size of only two, three, or four lines in diameter, I have frequently known to occasion little or no murmur. Osseous asperity, alone, without contraction, produces considerable murmur. From the above premises, it may be stated as a general principle, that the loudness of a murmur is in proportion, not only to the roughness of the obstacle, but also to the quantity of fluid transmitted through the valve and put in preternatural commotion by the obstacle. The effect we should naturally expect to be aided by the force and velocity with which the fluid is impelled; and, accordingly, we find that when the ventricle is hypertrophous, or the circulation hurried, the murmur is proportionably louder.* A simple illustration of these doctrines may be obtained by employing air instead of fluid, as the sonorous medium. Thus, if air be blown with equal velocity through a large, and a small orifice or tube, the sound is louder from the former. If the velocity be increased, the sound is proportionably augmented, and it is, cæteris paribus, always louder when the tube or orifice is rough or unequal.

A contraction of the mitral or tricuspid orifice to the size of the natural aortic or pulmonic orifice, generally produces a murmur. On what does this depend? Not on the mere circumstance of the contraction; for the aperture, being still as wide as that which affords egress to the blood, ought, therefore, to be equally capable of admitting the fluid without murmur. The murmur depends, I imagine, on the different configuration and position of the arterial, and the auriculo-ventricular orifices. The latter, instead of opening into tubes of their own dimensions, as in the case of the aortic and pulmonary orifices, open, generally with more or less infundibuliform projection, into spacious cavities; and, as the blood, on its delivery, must diverge to fill these cavities, it forms, in this act, various whirls, eddies, and counter-currents, which I conceive to be the cause of the sound. When the contracted valve, instead of projecting into the ventricle, is stretched tense across its orifice, the murmur is proportionably louder. This probably depends on the hydraulic law, that fluids escape with a more abundant and tranquil stream from a convergent pipe, than from a simple perforation of the same dimensions, through a plane surface. It is, perhaps, partly for an analogous reason that a *slight* patescence of the mitral or tricuspid valve occasions, by regurgitation, a louder sound than might be anticipated from the smallness of the aperture; for, in this case, from the construction of the valve, the discharging orifice projects into the fluid to be discharged,—a circumstance the most adverse of all others to the free and tranquil delivery of the fluid.* This, however, is not the

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* While this sheet is passing through the press, I find that Dr. Williams had already conjectured that murmurs in the heart and arteries were occasioned by "the motion of liquids in, or against, solids of a particular form." Rat. Expos. p. 50.

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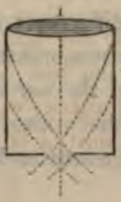
* Venturi found, that any vessel or reservoir discharged less through a simple, circular hole in its base; Fig. 1, than through one, to which was affixed, a short tube, of the same diameter as the


Discharged
62 quarts
in 100
seconds.

only reason for the loudness of the murmur. Another is, that, in consequence of the superior power of the ventricular contraction, the blood, propelled retrograde by it, moves with greater force and

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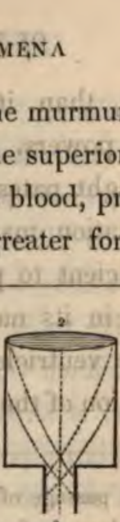

hole, and twice the length of its diameter. Fig. 2. He found, again, that, if the tube was pushed up some distance into the vessel, as Fig. 3. the flow of water was diminished, even to less than issued from the simple aperture.

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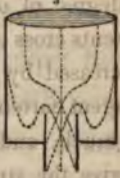

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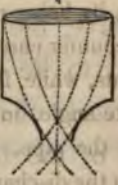

Sir Isaac Newton had previously ascertained, that fluid, tending from all parts of a reservoir to one common centre or orifice in the bottom, proceeded in curves; and that the solid figure, represented by these curves, was an hyperboloid of the fourth order. It occurred to Venturi that, such being the natural form in which water tends to discharge itself, a pipe of that form, would favour the discharge: and accordingly he found this to be the case. Fig. 4. Conceiving, further, that the curve in which water naturally tends to an orifice, was, from the inertia of water, continued beyond the point of discharge, he made the pipe trumpet-mouthed beyond its narrowest point, in the same curve as before it; and from this he obtained the maximum discharge. Fig 5.

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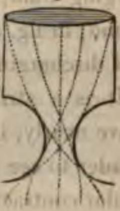

These differences in the quantity of discharge from orifices of the same area, depend on the currents crossing each other at the orifice, and thus constituting a greater or less obstruction to velocity, than it flows forwards by its natural moving powers.

A slight patescence of the valve admitting of regurgitation, may result from a structural lesion not sufficient to present an obstacle to the blood flowing in its natural direction from the auricle into the ventricle,—as that, for instance, from a contraction of the chordæ tendineæ, preventing the

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the direct passage of the whole body of fluid. It is obvious that the degree of obstruction will be greater in proportion as the currents cross at greater angles, and that it will be still further increased by any counter-currents or eddies opposing the convergent currents. Thus, in fig. 1. the currents of fluid converge much, and, by their contending influence when crossing, give the stream a twisted or screw-like form. In fig. 2. the currents converge less, because their divergence is limited by the pipe, and the convergence and divergence are, from the inertia of water, in the direct ratio of each other. In fig. 3. the currents descending from near the outside of the vessel, by being reflected up to reach the discharging orifice form counter currents which oppose the descent of the central currents, while, further, they are themselves forced to turn at an acute angle, in opposition to their inertia, before they can enter the pipe. Hence the obstruction is even greater than when the discharging orifice is a simple perforation. In fig. 4. the form approximates very nearly to the natural form of discharging fluid, whence the discharge is proportionably greater; and, in fig. 5. where the form is identically the same as that of discharging fluid, the maximum discharge is obtained. It is a striking fact that the auriculo-ventricular orifices have nearly, if not exactly this form. It will be easy for the reader to see the application of the figures to the various valvular contractions.

margins of the valve from coming in perfect apposition: and in this way is to be explained a phenomenon which I have frequently noticed; namely, that a murmur from regurgitation sometimes accompanies the first sound, when none attends the second. (Case of Dennis). A slight contraction, indeed, such as, for example, to diminish the circumference by a quarter, or from that to half an inch, does not, unless accompanied with ruggedness, occasion any appreciable murmur with the second sound; for the blood has little sufficient space to pass with tranquillity; but if, at the same time, the ventricle is dilated and hypertrophous, a considerable murmur may be created; for the increase of the quantity and velocity of the blood which enters from the auricle, produces the same effect as a greater degree of contraction of the orifice.

III. *The Mechanism of morbid Murmurs from Hypertrophy with Dilatation*, 63. *Not occasioned solely by the increased Quantity and Force of the Blood expelled*, 63; *but also by the altered Direction of the Currents*, 63.

In a case which presented itself to me in 1825,* I was led to notice that murmur was produced by a disproportion between the cavities and the orifices, consequent on enlargement of the former. I have just shown in what manner the disproportion occasions murmur of the second sound: it remains to be explained how it produces the same effect on the first,—an effect of frequent occurrence in cases of great hypertrophy with dilatation. It is, no doubt, partly attributable to the increased quantity and force of the blood expelled by the ventricular systole: but this is not all; for the same increase may take place without causing murmur, when, in a healthy individual, the circulation is simply accelerated. Supposing, then, as much blood to be expelled at each systole, through an orifice of given dimensions, by an enlarged ventricle acting calmly, as by a natural sized one excited to increased action, if the latter propels its contents without, and the former with murmur, it follows that this phenomenon is dependent, not on the force and quantity of the blood expelled, but on some other circumstance which alters the manner in which the fluid moves. This circumstance appears to me to be, the changed form of the ventricle; for as, in hypertrophy with dilatation, the cavity is more spherical than natural, and its artery consequently rises more abruptly with respect to its internal surface, the currents of blood reflected from its sides meet in the orifice at more obtuse angles,*

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* Vid. Lond. Med. Gaz. Sept. 5, 1829, p. 420.

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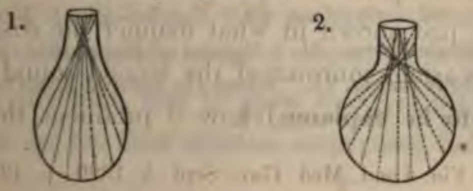
* 1. A natural shaped ventricle. 2. A rounded one.



and thus, by their collision, not only give rise to the murmur, but impede each other's passage into the vessel. For the latter reason, the pulse is sometimes small and weak, when the impulse of the heart is violent,—a paradox with which authors have been much perplexed.

IV. *The Mechanism of Murmur of the Heart and Arteries independent of organic Disease*, 65. *Circumstances under which it occurs*, 65. *Laennec's Account inconsistent*, 66. *Spasmodic muscular Contraction will not account for it*, 67. *Purring Tremor of Arteries inexplicable to Laennec*, 68. *Attributed by him to nervous Action*, 68. *Author's Account of the Phenomena*, 69. *Modified Motion of the Fluid their cause*, 69. *Proofs.* 1st. *Experiments, showing that Fluids moving through Pipes, produce Murmur*, 70. *Friction its Cause*, 71. 2nd. *When there is Murmur, there is modified Motion of the Blood to account for it*, 71. *Experiments by loss of Blood, on Dogs*, 72. *Jerking Impulse and Pulse with Murmur and Thrill produced*, 72. *Connected with increased Velocity of the Circulation*, 74. *Also with Attenuation of the Blood*, 75. *This exists in most nervous Persons subject to Murmur*, 75. 3rd. *Author's Explanation applies under all Circumstances*, 75.—*In Perecarditis and Adhesion of the Pericardium*, 75.—*In reaction from loss of Blood*, 76.—*In nervous, hysterical, and hypochondriacal Subjects*, 76. *Partial arterial Pulsation explained*, 78. *Inflammatory Pulse not attended with Jerking or Murmur*, 79. *First Principle of the Heart's Action*, 80. *Opinions*, 80. *Cerebro-spinal Nerves*, 80. *Ganglionic Nerves*, 81. *Opinion of Mayo, that the motive Faculty is innate*, 81. *Blood the natural Stimulus which regulates its Movements*, 82.

Before proceeding to assign the cause of this phenomenon, it is necessary to be agreed as to the circumstances under which it occurs. The account which Laennec gives of it, and of the concomitant phenomena purring tremor (*frémissement cataire,*) and (what I conceive to be merely a less degree of the same) thrilling (frémissement) of the arteries, does not accord with my own observation, and it appears to involve several inconsistencies, which render the phenomena equally inexplicable on his own and on every other theory. To question any thing which Laennec explicitly states as a fact, is hazardous: the more I have studied his works, the more have I become sensible of this, and felt astonished at the wonderful accuracy of his powers of observation. With respect to the subject before us, however, it is both apparent from the statements in his treatise, and well known to those who were acquainted with him, that he had not satisfied his own mind: that he was conscious of incongruities which he could not reconcile, and of difficulties which he was unable to surmount. With less presumption, therefore, may I enter on an investigation which his genius can only be said to have left incomplete; and I do it with more satisfaction, as I have to advocate the cause of auscultation against its great inventor, and to show that the doctrines broached in his first edition respecting bellows-murmur as a sign of valvular disease, were not, as he imagined, invalidated by the more extended knowledge of the nature of this phenomenon which he supposed himself to have acquired at a later period.

"The bellows-murmur," says Laennec, "may accompany the *diastole* of the heart and that of the arteries, and it is connected with them in such a manner as to replace and entirely annihilate their natural sound; so that, at each diastole, the ventricle, the auricle, or the artery in which the phenomenon takes place, yields a distinct sound of a puff of the bellows, the noise of which ceases during the systole." (De l'Auscult. t. ii. p. 422.)

This account is clearly inconsistent with itself. It is certain that the murmur in question takes place synchronously in the heart and arteries: it cannot, therefore, take place during the *diastole* of both, as the diastole of the one coincides with the systole of the other. The error consists in saying that it coincides with the diastole of the cavities of the heart. It is not possible, for instance, that a murmur of the ventricular diastole could replace or destroy the natural sound of its systole, because the two sounds are consecutive—not simultaneous. Nor is it possible that the auricular diastole could occasion murmur; for we have seen in the antecedent experiments, that this motion has not sufficient latitude to occasion any sound whatever. Granting, for a moment, that the murmur does, as Laennec imagines, accompany the diastolic movements of the heart, this view is irreconcileable with his explanation of the cause of the phenomena; for, having disavowed his belief that the cause is connected with the motions of the fluid,* he says, "The perfect similitude of the intermittent *muscular sound* (bruit musculaire) and of the bellows-murmur of the heart and arteries, appears to me entirely to decide the questions which I have above proposed on the nature of this murmur, and to prove that it is referable to a real spasmodic *contraction*, whether of the heart or of the arteries. The possibility of a spasm of the heart needs not be demonstrated, since that organ is muscular. With respect to the arteries, the circular fibres which compose their middle coat appear to announce a tissue endued with the faculty of contraction."† Now, if spasmodic *contraction* be the cause of the bellows-murmur, this murmur cannot take place during the diastole of the heart, which, according to the best authorities, is an act of relaxation.

Neither will spasmodic contraction account for the bellows-murmur in the arteries; for the murmur takes place during their diastole, not during their systole. Laennec, apparently conscious of this inconsistency, endeavours to reconcile it by saying that the murmur occurs while the artery is in the act of turning from its diastolic, to its systolic state. There is, however, no doubt that it occurs while the artery is in the progress of dilatation.

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* De l'Auscult. tom. ii. p. 429.

† Ibid. p. 440.

Respecting the purring tremor of arteries, Laennec avows that, notwithstanding all the pains he has taken for the purpose, he has not been able to discover any satisfactory reason for the phenomenon.* Nor is this surprising: for having attributed the twin phenomenon, bellows-murmur, to spasm,—a state tending to place an artery in a state of constriction and immobility, rather than of vibration, he has no other physical resource remaining, by which to explain the purring tremor. He accordingly yields to the difficulty; for it is little more than a substitution of words for ideas, to say, "it is at least extremely probable that the purring tremor depends upon a particular modification of the nervous action;"† (innervation) and that "the three phenomena, bellows-murmur, purring tremor, and the thrilling pulse, are attributable to different, though analogous, modifications of the action of the arteries and the heart, and that the one cannot be regarded as a more or less intense degree of the other."*

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* De l'Auscult. tom. ii. p. 452.

† Ibid. p. 453.

Having thus endeavoured to present a brief sketch of a subject, which, from its obscurity, has in general occupied several chapters, I proceed to offer an explanation of the murmurs in question on a different principle.

As my own experience does not accord with that of Laennec as to which motions of the heart are accompanied by the murmur, it is necessary to premise that I have found it accompany the *systole* of the ventricles; and not the diastole, unless it, at the same time, and in a predominant degree, attended the systole. In the arteries, it coincides with their diastole. The purring tremor occurs at the same moment and is a result of the same cause. The arterial thrill is nothing more than a less degree of the purring tremor.

Both by experimental and pathological evidence, I am led to believe that the murmurs and tremors, as well in the heart as in the arteries, are occasioned by modifications in the motion of the fluid. To establish this point, it is necessary to prove, 1. that liquids permeating tubes, do occasion sound; 2. that modifications calculated to elicit the sounds, do take place under the circumstances in which the sounds occur. 3. That the explanation applies equally, whatever be the circumstances under which the sounds occur.

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* De l'Auscult. tom. ii. p. 767.

1. That a bellows sound is produced by the transmission of a fluid, without any intermixture of air, through a tube, though questioned by some, is a fact too easy of demonstration to require discussion. Having just returned from a repetition of the experiment,—one which I have frequently performed, I find the rushing murmur so distinct and close to the ear, as to preclude the idea of a fallacy from the movement of a piston or any other cause: I find the sound to vary in intensity according to the velocity with which the fluid is propelled, to be increased by bending the tube at an angle, and to be still further increased, but also modified, by the admission of air; becoming of a rattling nature, totally different from any sound heard in the heart or arteries. What experiment proves, the principles of hydraulics would lead us to anticipate. It is a fact, established by the investigations of Newton, De Buat, Bernoulli d'Alembert, Robison, Venturi, Dr. Young, and others, that the progress of fluid through pipes is retarded by friction against their interior; and that the retardation is increased by all projections, irregularities, and sudden bends; for the fluid,

striking against these, forms reverberations and eddies, which impede its current as effectually as solid obstacles. The friction increases with the increase of velocity, and, beyond a certain point, it increases in a much more than simple ratio. Thus, if a steam engine of ten horse power will propel a vessel ten miles an hour, one of a hundred would not suffice to propel it twenty. Now the friction,—in other words, the collision of the particles of fluid against the sides of the vessel and against each other, by producing vibrations of a certain rate of rapidity, is the cause of the sound; and these two phenomena, the friction and the murmur, are, consequently, in the direct ratio of each other. Hence it appears that the murmur is produced in strict conformity with the general axiom, that the particles of all bodies, when thrown into sufficient vibration, generate sound.

2. It is next to be proved that, when the sounds in the heart and arteries do occur, there is an increase of friction, dependent on a modification of the motion of the blood, to account for them.

Being engaged with Dr. Marshal Hall in a series of experiments on the effects of loss of blood, &c. a subject for the elucidation of which the profession is deeply indebted to that eminently sagacious observer, we took the opportunity of studying the stethoscopic phenomena of the circulation under all the circumstances of collapse, reaction, &c.

Eight or ten dogs were blooded more or less frequently, from once to ten or twelve times, and at intervals varying from twenty-four to seventy-two hours. The results were, that, on the day following the first or second abstraction of blood to the amount of eight or ten ounces, the systolic sound of the heart, previously loud and clear, became attended with a whizzing or sawing murmur,* the impulse increased and became unusually smart or abrupt, and the pulse became quick and jerking, with a thrill and a throbbing, perceptible over the whole body. These phenomena increased up to the fourth or fifth bleeding, when they appeared to attain their maximum, the sawing sound being extremely loud, the impulse and pulse violently jerking and bounding, the arterial thrill or purring tremor excessive, and the throbbing perceptible not only when the finger was placed on an artery, but when the hand grasped a large surface of the body. A hissing bellows-murmur was, moreover, distinctly heard, when the stethoscope was placed over any considerable artery, as the femoral or carotid. The pulse at this time generally beat from 150 to 190 per minute, its natural standard being about 120.

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* These, it will be understood, are merely varieties of the *bellows-murmur*,—a term which may be taken in a generic sense.

The phenomena underwent the following changes in correspondence with changes in the circumstances. The animals being extremely nervous and irritable, the pulse was instantly accelerated ten or fifteen beats per minute by the slightest excitement, as that of being moved or startled; and the murmur and jerk sustained, in consequence, a remarkable increase.

After reiterated venesections the pulse became small and weak; but, so long as it remained jerking (or *sharp*, as some would now designate it) the murmur continued, though not so loud as previously.

If venesection was omitted for three or four days, reaction subsided; and in proportion as the pulse and impulse became softer, though without a loss of *real* strength and fulness, the murmur, both of the heart and arteries, the purring tremor, the general throbbing, and the nervous irritability, gradually disappeared.

If, during the full prevalence of all the phenomena, the animal was bled to the approach of syncope, the pulse and beats of the heart, reduced to about 100 per minute, became feeble and soft, and at the same time lost all murmur and thrill;

but, in the course of from fifteen to thirty minutes, reaction was re-established and all the symptoms recurred.

If the animal was held erect by the fore legs, a posture which, either by diminishing the afflux of blood to the brain, or by obstructing the circulation through the heart and lungs, caused the gradual supervention of syncope, the pulse became slow, soft and feeble, and the murmur and thrill were suspended; but they were promptly restored to their former state when the animal was placed on its legs.

From these experiments it may be concluded that the murmurs and tremors are dependent on the spasmodic abruptness of the heart's contraction, or, more rigidly speaking, on the velocity with which the blood is propelled in consequence of that abruptness:—a velocity which implies an augmentation of friction, and consequently of arterial vibration. That an increase of velocity alone suffices to excite the phenomena, independent of an increase of *real* force of the heart's contraction, is proved by their existing when the pulse was small and weak, provided it was jerking or sharp. I conceive the primary cause to reside in the heart and not in the arteries, because the action of the latter was always in exact proportion to that of the former. Some exceptions to this, which have been observed in the human subject, I shall presently explain. A collateral circumstance, which may probably favour the production of murmur and tremor, is, the attenuation of the blood: for its particles, having lost a proportion of their lubricity, are better calculated for rapid motion, and consequently for the production of murmur and vibration by the collision against each other and against the walls of the containing vessel. This attenuation (the existence of which was strikingly displayed in some of the above experiments by the crassamentum being reduced to a very small fraction,—one sixth to one twelfth for instance, of the serum) is not confined to cases of excessive loss of blood, but exists also in a large proportion of the nervous cases which present bellows-murmur. We now come to the third branch of the subject; namely, to prove—

3. That the explanation applies equally, whatever be the circumstances under which the sounds occur. In order to substantiate this point, it is merely necessary to show that, where the sounds *do* occur, they are attended, to a greater or less degree, with a jerking action of the heart and arteries analogous to that which has been described. Pericarditis and adhesion of the pericardium,

affections remarkable for a bellows murmur hitherto not satisfactorily explained, will, in their proper articles, be proved to fall under these conditions.

Another class in which the bellows-murmur prevails, consists of those who are under the influence of reaction from excessive loss of blood, whether by the lancet or by hæmorrhage. The jerk and throb of the pulse, even when small and compressible, is well known to all who have witnessed a case of uterine hæmorrhage. In cases of active hæmorrhage, as hemoptysis and even epistasus, I have known the throb to supervene sooner than could be accounted for by the quantity of blood lost. In these cases it appears to be attributable either to the same plethoric habit and irritable temperament which predisposed to the hæmorrhage, or to the fright which seldom fails to be occasioned by the unexpected appearance of blood.

The remaining and by far the largest class of individuals in whom the bellows-murmur is apt to occur, consists of young people of plethoric habit and delicate, irritable temperament, subject to hysterical and hypochondriacal affections, and to nervous palpitation. In such, the murmur may come on whenever the circulation is excited, and, for exciting it, the most trivial causes are sufficient. I have seen a single cough, or a full inspiration, or a little flatulence, produce the effect for a few beats only; while the act of turning in bed, of rising suddenly, of being startled by any noise, has occasioned it for several minutes. An emotion of grief or pleasure will sometimes produce a more considerable and permanent effect. I have often been assured by patients that the momentary flash of an idea across the mind, has sufficed instantly to excite a violent fit of palpitation, and that this has recurred several times a day, whenever the same idea has presented itself. That, under so irritable a state of the nervous system, the heart should contract with spasmodic abruptness, might be anticipated. What theory points out, experience proves; for the sharp, jerking pulse and beat of the heart of a patient in a state of nervous agitation, is too well known to require demonstration. Sometimes, when the nervous excitement is excessive, a violent throbbing is perceptible over the whole body, and the bellows-murmur and thrill are distinct in every considerable arterial trunk. When such is the case, the anxiety and distress of the patient are extreme, and his situation is not exempt from danger. Though death is rare when the symptoms are purely nervous, I have once seen it occur. The patient, a healthy plethoric young female, who became affected on the intelligence that her husband had deserted her, lay twenty-four hours in a state of almost complete insensibility, and the violently bounding, jerking and thrilling arterial throb, together with universal flushing, heat and perspiration of the surface, resisted every remedy, and only subsided with the wane of life.

A few apparent anomalies remain to be explained. One portion of an artery, as the abdominal aorta, or the radial in a case of whitlow, may pulsate more strongly than another. The most simple and generally received explanation of this, is, that arteries are irritable parts, which, under the circumstances alluded to, and under many others, as blushing, increased local determination for the purpose of nutrition and secretion—exemplified by the state of the uterus and mamma during pregnancy, become *relaxed* or lose a portion of that tonic power by which they resist and support the distensive force of the heart's contractions. Consequently, as liquids flow most readily in that direction which offers the least resistance, the relaxed vessel admits an increased quantity of blood at each contraction of the heart; hence its throb,—which is a passive—not an active movement; and, as the augmented afflux is necessarily accompanied with increased velocity of the current, at least until the vessels become gorged, the result is an increase of friction, producing murmur. Another circumstance contributes to the production of the murmur: the relaxed artery, by yielding to the pressure of the heart, becomes not only distended, but elongated and tortuous. Hence, according to the principle developed in page 70, the friction and murmur are increased. One of the most tortuous arteries is the placental during gestation, and in none is the bellows-murmur more distinct. Some have imagined that the pulsation, in these cases, is quicker than that of the heart. It is scarcely necessary to say that this is an error. Corresponding arteries in different limbs have been said to beat alternately stronger than each other,—to me, when I could persuade myself of the fact, it has always appeared to depend on differences of posture, by which the free afflux of blood to the one limb or the other, was prevented. In all circumstances, however, the murmur and thrill, according to the explanation which I have offered, depend on the motion of the fluid.

It may be remarked, in conclusion, that the purely inflammatory pulse, though more or less quick, full, strong, and hard, is not jerking nor attended with bellows-murmur. These distinctions, which, to the inexperienced, may appear refined in description, are perfectly familiar to practical men; and it is of great importance to the young practitioner that he make himself intimately acquainted with them, as such knowledge will not only facilitate his diagnosis, but prevent the unnecessary and often, in nervous cases, pernicious abstraction of blood for imaginary fever or inflammation.

After having studied all the phenomena, both physiological and pathological, of the heart's action, an ulterior question naturally presents itself —what is the first principle,—the primary spring, which gives motion to the great organ of the circulation. As this is rather a question of physiological interest, than one, the determination of which is essential to the present subject, I shall merely glance at the existing opinions, and leave the reader to prosecute the inquiry by referring to original sources of information.

It is the persuasion of many distinguished physiologists, particularly those of the French school, founded, as they conceive, on experiment and observation, that the nerves of the heart constitute its motive principle. But, as these nerves are derived from two sources,—the cerebro-spinal, and the gonglionic systems, it was a question which of the two were destined to impart the faculty of motion, Le Gallois ascribed this faculty to the spinal nerves; but his conclusions were invalidated by the researches of Lallemand, who found that the heart beat in the fœtus though destitute of spinal marrow; and by the experiments of Wilson, Philip, Mayo, Clift, and many others, who found that the action of the heart survived the destruction of the spinal marrow, and even the excision of the organ out of the body. Hence it appeared to result, that, while the cerebro-spinal nerves or par vagum, according to the brilliant discoveries of Mr. Bell, connected the heart with the lungs, the stomach, the thoracic muscles, the face,—with all the parts, in short, associated in the functions of respiration and expression; the ganglionic nerves or, in other words, the great sympathetic, was the principle which imparted the faculty of motion. Thus it was explained how the action of the heart was independent of the will, while it was strongly under the empire of the passions and of corporeal nervous sympathies.

Mr. Mayo, on the contrary, founding his opinion on a train of profound and ingenious reasoning, partly developed in his work on physiology, but which he has done me the favour to explain more at length, entertains the belief that the motive principle of the heart is an innate power independent of the nerves; and that, while it is the *natural state* of voluntary muscles, both in the living body and before the loss of irritability after death, to remain relaxed, unless excited by special impressions; it is, on the other hand, the *natural state* of the heart, an involuntary muscle, under the same circumstances, to contract and dilate alternately for a time, in the absence of external impressions. In the turtle, an extremely vivacious animal, the alternate actions continue for a very long period. I have seen them last for upwards of an hour, though sections were made both longitudinally and transversely into the cavity of the ventricle. The motive principle, whatever it be, appears to be more or less exhausted by each contraction; for a puncture, made immediately after the effort, does not cause a repetition of it; but, made at the interval of a few seconds, it produces the effect.

Whether the motive principle be nervous, or an innate power, it is sufficient for our present purpose that the organ replies to a stimulus. This stimulus, in the natural state, is the blood; and, by flowing into the heart in suitable quantities at definite intervals, according to the principles above explained, it appears to maintain the action of the organ in a state of regularity.

PART II.

INFLAMMATORY AFFECTIONS OF THE HEART AND GREAT VESSELS.

THIS Part will be divided into three chapters. The first will be devoted to inflammation of the external membrane, (pericarditis); the second, to that of the muscular substance, (carditis); and the third will comprise inflammation of the internal membrane both of the heart and of the arteries, together with the inflammatory diseases of these vessels in general. As the internal membranes of the heart and the great vessels are continuous, and their affections almost identical, it would be an unnecessary multiplication of distinctions to treat of them separately. The connection subsisting between inflammation of the external, and of the internal membrane of the heart, will be pointed out in pericarditis.

CHAPTER I.

ON PERICARDITIS.

SECTION I.

ANATOMICAL CHARACTERS OF PERICARDITIS.

ACUTE. 1*st.* Preternatural redness of the Pericardium, 85. *Not a positive Proof of Inflammation,* 85. 2*d.* Coagulable Lymph adhering to the Pericardium, 86. *Its Colour, alveolar Appearance, &c.* 87. *Use of Lymph,* 88. *Adhesion how a Process of Reparation,* 88. *Causes preventing it,* 89. *Description of the Process,* 90. *Recent—old,* 90. *Thick and laminated Adhesions,* 91. *Purulent—indurated,* 91. *Partial Adhesions,* 91. *Loose—close,* 92. *Forming Abscesses around the Heart,* 92. *Partial Pericarditis, rare—terminates in recovery,* 92. *White spots on the Heart,* 92. *Granulations,* 93. 3*d.* Fluid effused within the Pericardium, 93. *Its Colour—Transparency,* 93. *Quantity great at first—quickly absorbed,* 93. *When secreted by Lymph, it becomes turbid, purulent, sanguinolent,* 94. *Cause of Sanguinolency,* 94. CHRONIC. *Its Characters differ little from those of the advanced Stages of the acute,* 95. *Intimate Adhesion, caused both by Acute and Chronic,* 95.

THE anatomical characters of acute inflammation of the pericardium are, 1. preternatural redness of the membrane; 2. coagulable lymph adhering to its surface; and 3. fluid effused within its cavity. They will be treated of in succession, and at some length. For, as the anatomical characters are the key to the symptoms, the latter cannot be understood, and, consequently, so rapid and fatal a malady cannot be treated with the promptitude and decision essential to the safety of the patient, unless the characters in question, and their intimate connection with the symptoms, are thoroughly known to the practitioner. To this subject, therefore, I would particularly direct the attention of the student.

1. *Preternatural redness of the Pericardium.*— The redness very seldom pervades the whole of the inflamed portion. It presents itself sometimes in numerous small scarlet specks with a natural colour of the intervening membrane, sometimes in spots of greater or less magnitude formed by the agglomeration of the specks, and sometimes in patches of considerable extent. Even these, however, have, almost without exception, a dotted or mottled character. In a drawing before me, which I made from a case of very acute and rapid pericarditis, nearly the whole of the reflected membrane, underneath a layer of soft, primrose-coloured lymph, is of a vivid, mottled and dotted red. In some cases, according to Laennec, though the inflammation, judging of it by the thickness of

the false membranes, had been very severe, scarcely any redness exists. Such is the case, with respect to the surface of the heart, in the drawing to which I refer. Vascular injection of an arborescent and sometimes stellated appearance, accompanies redness, and is generally proportionate to it in degree. Occasionally, however, the redness is uniform like a stain.

The pericardium is very rarely, if ever, thickened; that which is often regarded as thickening, being nothing more than superimposed and intimately adherent false membrane.

When acute pericarditis degenerates into chronic, the redness loses its brilliancy, sometimes becoming very deep and of a brownish colour, and sometimes acquiring a cinnamon hue. Beneath a layer of lymph of this colour, I have seen the surface of the heart of a bluish white appearance, like the spots so frequently found on this organ.

Redness *alone* does not afford conclusive evidence of pericarditis, as all serous as well as mucous membranes are liable to vascular injection from various causes independent of inflammation. To afford such evidence, therefore, the redness must be conjoined with an effusion of lymph or sero-purulent fluid.

2. *Coagulable lymph adhering to the surface of the pericardium.*—The inflamed pericardium

secretes serum and lymph conjointly, and in a fluid state, from the same vessels. Soon after the secretion has taken place, the lymph concretes, while the serum remains fluid. The former, when recent, is of a pale straw-colour, and of a soft, tender consistence, becoming firmer and more tenacious as it grows older. Though occasionally deposited in detached lumps and spots, it generally forms continuous layers, sometimes covering a portion only, but more commonly the whole, or nearly the whole, of the pericardium. The thickness of the deposition may vary from a line to an inch; but, from a line and a half to three lines is its ordinary mean. Its adherent surface is smooth; the opposite is rough and singularly figured. In drawings before me, where I have delineated from nature all the appearances that I have ever witnessed, the free surface is sometimes pitted with small depressions at tolerably regular intervals, presenting the aspect of a fine reticulation or of the section of a sponge. This occurs principally where the layer is thin; where it is thick, the surface is distributed into more spacious cells, often as large as a pea, and separated by coarser partitions. The partitions are sometimes irregular, being higher and thicker in one part than another; in which case the effect exactly resembles that produced by separating two flat plates, between the surfaces of which a layer of soft butter has been spread. At other times the partitions are very regular; in which case, the appearance, as Corvisart observes, is analogous to that of the second stomach of a calf. Occasionally they are very thick and rounded, and then they have an appearance somewhat similar to that of a congeries of small earth worms. Not unfrequently they are shaggy and flocculent, hanging in shreds like tow. In one drawing, from a case which had become chronic, no cells are apparent, but the lymph is arranged in transverse, and, as it were, plaited wrinkles, like undulations of sand on the sea shore. When lymph becomes old, and not adherent, it acquires a deeper hue, varying from cinnamon to an intense brown-red or mahogany colour. When of the latter colour, it usually secretes bloody fluid.

Now, what is the object which nature proposed to herself in the effusion of lymph? Unquestionably to effect reparation:—the object for which the effusion is designed in whatever part of the system it takes place. But how, it may be inquired, can it effect reparation in the pericardium? By causing adhesion. Supposing that the inflammatory process does not terminate by resolution—by the complete absorption of both lymph and serum, the most desirable termination which remains is adhesion; for, should this not take place, the lymph becomes a secreting surface, which effuses more and more lymph and serum, until, in a short time, the cavity is completely distended, and the action of the heart so embarrassed that a fatal termination speedily ensues. But, should adhesion of the opposite surfaces take place, by which further effusion is prevented, life may be prolonged for a considerable period—even for years; though, as will presently be explained, the adhesion, so far from being a *perfect* reparation, gives rise to another form of organic disease, which eventually proves destructive to the patient.

Adhesion takes place in some cases and not in others,—a circumstance which has been attributed to a difference in the quality of the lymph, dependent on the greater or less energy of the inflammation, and in consequence of which it possesses different degrees of aptitude for adhesion. This explanation, though perhaps not unsound, is scarcely applicable to the pericardium; for, here, the union or non-union depends merely on the absence or presence of fluid in the cavity; the best lymph, equally with the worst, being incapable of uniting when interposed fluid prevents the apposition of the opposite surface. Hence it is that the upper part of the pericardium, where it is reflected from the great vessels, often adheres, when the lower part does not: and for the same reason it is, that, when the whole of the peritoneum is covered with lymph, the intestines adhere to each other, but their adhesion with the walls of the abdomen is prevented by the interposition of fluid.

Hence the immense importance in pericarditis, of prompt and energetic treatment in the first instance, in order, if resolution cannot be effected, to cause absorption of the fluid and thus afford the opportunity for adhesion. Temporizing indecision is inadmissible; for unless one or other of these terminations be induced, the patient inevitably dies.

Such is the object of adhesion: we have now to describe the process. When the fluid has been sufficiently absorbed, the depositions of lymph on the opposite surfaces of the pericardium come in contact, thicken, blend, and gradually become organized by vessels presenting themselves under the successive appearances of blood-stains, straggling lines, and, lastly, of uniform pinkish vascularity, susceptible of injection from the pericardium. The depositions are thus converted into perfect cellular tissue, by which the contiguous parts are more or less firmly, closely, and extensively agglutinated. When adhesion is of recent standing, the lymph is generally thick, and separable by mere tearing into two layers, one adhering to each fold of the pericardium. In proportion as the disease is older, the false membrane is thinner and firmer, consisting, in cases that date several years back, of the finest layer of dense cellular tissue. In some, even this is not perceptible, the folds of the pericardium having become amalgamated—apparently without the intervention of any membrane, so as with difficulty to be separable, even by the scalpel. (Case of May, and a Boy.) It is in such cases that pathologists have sometimes erroneously supposed the heart to be destitute of a pericardium.

In some rather protracted cases, generally of at least two or three months duration, where, though adhesion has been established, inflammation has either recurred or never been completely subdued; an additional interstitial deposition of lymph takes place, which has been known to thicken the adventitious mass to the extent of an inch and upwards. In this case it sometimes possesses a laminated texture, the layers of which are progressively redder in proportion as they are nearer the heart; and sometimes it exhibits different degrees of consistence in different parts, one being almost liquid and purulent, while another has the density of tubercular induration.* Such cases are ordinarily fatal at no very remote period.

But adhesions are not always universal; in cases of partial pericarditis they are restricted to the portions inflamed; and when these portions are limited, the adhesions are not close or intimate; for, as the gliding motion of the heart within the pericardium is not prevented, it stretches the adherent lymph, and converts it into long loose bands of cellular tissue. But when the portions inflamed are extensive, partial adhesions are sometimes close and firm, and the intervening parts of the pericardium may be healthy and in contact. Instances occasionally occur of adhesions being partial though the inflammation had been universal; and the parts not united are overspread with lymph and separated by purulent fluid, thus constituting a series of small, detached abscesses around the heart.

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* Latham Lond. Med. Gaz. Vol. iii. p. 5.

Cases of partial pericarditis, (exclusive of white spots, of which we shall presently speak) are very rare in proportion to those of general, scarcely amounting, according to Laennec, to one in ten.* They almost always terminate in recovery, and the adhesions, if loose and long, seldom lead to enlargement of the heart.

Partial pericarditis sometimes leaves no other vestiges than opake white or milky spots, which are a well known appearance on the surface of the heart. They vary in extent from a few lines to two or three inches in diameter; their thickness is about that of the nail: they consist of condensed cellular tissue, and, with a little care, they may generally be detached without injury to the pericardium beneath. This membrane is commonly somewhat injected, though not thickened.

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* De l'Auscult. tom. iii. p. 655.

Sometimes lymph is converted into small, roundish, soft granulations like concrete albumen, with which the pericardium is more or less extensively studded.

3. *Fluid effused within the cavity of the pericardium.*—It has been stated that serum is effused conjointly with lymph, from the vessels of the inflamed pericardium. This fluid is sometimes transparent and either of a faint yellow more or less tinged with green, as that of the interior of a lemon, or of a pale fawn colour; at other times it is less transparent, but very seldom milky or opake from containing particles, filaments, or flakes of imperfectly concrete albumen. Its quantity, though variable, is in general considerable at the commencement, that is, within the first two, three or four days, of the disease,—not unfrequently amounting to more than a pint. Corvisart once found four. It is speedily diminished, however, by absorption when the first violence of the inflammation begins to subside; and, after the lapse of a few days, it is, in the majority of cases, not more abundant than

the concomitant exudation of lymph. Sometimes, indeed,—even in very acute inflammation, the absorption is so complete that no serum whatever is found, while a copious exudation of thick, concrete lymph fills and agglutinates the whole cavity.

Should complete absorption of both the fluid and lymph not take place, nor yet adhesion of the pericardium be established, the fluid presently assumes a very different character. For it is no longer secreted by the pericardium, but by the lymph itself, which, when organized, becomes a secreting surface; and its secretion, though at first consisting of clear serum, gradually becomes more and more turbid, milky and opake, until it eventually assumes the character of pus. Rarely, however, is perfect pus found in the pericardium;—probably because the patient dies from irritation before the suppurative process is fully established. Not unfrequently the fluid is bloody, (Cases of Porter and Snowden.) and the lymph of a red colour. (Case of Porter.) This is attributable to the tenderness of all newly organized structures, in consequence of which they are apt to become congested and to effuse blood when subjected to any unusual irritation or excitement. The excitement in the present instance, most probably consists in inflammation of the adventitious membrane, either renewed, or never wholly suspended.

Compression exercised by fluid, sometimes reduces the volume of the heart, and renders it, as it were, atrophous.

Such are the anatomical characters of acute pericarditis, both in its early and its advanced stages. It remains for me to make a few remarks on that form of pericarditis which appears, from the mildness of the inflammatory symptoms, to have been chronic from the first. Its anatomical characters do not differ very materially from those exhibited by the advanced stages of the acute form. The inflammation always pervades the whole of the cavity; the redness is deeper and duller than in the acute affection; the albuminous false membranes are, in many cases, totally deficient; and when present, they are thin, soft and fragile, as if wasted by suppuration: finally, there is always a more or less abundant effusion of turbid, milky, and sometimes completely puriform fluid. *Intimate* adhesions of the pericardium to the heart may follow the absorption of this fluid; but M. Laennec does not appear to me to be borne out either by facts or by analogy, when he supposes that chronic pericarditis is the sole cause of intimate adhesion, and that the acute affection only gives rise to loose adhesion by more or less elongated bands. According to my experience, the latter is the more frequent cause of intimate adhesion. (Cases of

Copas, May, a Boy, and many others after acute rheumatism.)

The muscular substance of the heart is, in general, not affected by pericarditis; but sometimes it is rendered redder or paler, browner or yellower, harder, softer, or more lacerable, than natural. These changes result from inflammation propagated from the pericardium to the muscular substance. They will be more fully considered in the article Carditis.

In scrofulous and phthisical individuals, tubercles are sometimes developed in the false membranes of pericarditis, and according to Laennec, they may cause the acute to pass into the chronic state, as frequently happens in the case of pleuritic and peritoneal false membranes.

SECTION II.

SIGNS AND DIAGNOSIS OF PERICARDITIS.

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Symptoms facilitates the Diagnosis, 103. *Further Causes of Obscurity*, 103. *Absence of Pain and other characteristic Symptoms*, 103. *Signs on which the Diagnosis is then to be founded*, 104. *The most acute Cases not, as Corvisart thought, the most obscure*, 104. *How to be detected when imitating other Affections*, 105. *Diagnosis from Inflammations of the Lungs*, 106. *Signs of Amelioration*, 107. Physical Signs. Impulse *of the Heart, increased, abrupt*, 108. *Pulse jerking, thrilling*, 108. *Causes*, 109. *Causes of the Persistence of these Signs after the Inflammation has subsided*, 109. Sounds—*of Ventricular Systole, loud, with Bellows-murmur*, 110. *Murmur does not exist in every case*, 110. *Murmur of the second Sound*, 110. *Causes of the Murmurs*, 111. *Resonance of the Cardiac Region on Percussion*, 113. *Undulatory Impulse from Hydro-pericardium*, 113. CHRONIC. General Signs. *In what different from the acute*, 113. *More obscure—Means of Diagnosis*, 114. Physical Signs. *Impulse, Sounds, undulatory Impulse, &c.* 115.

General Signs.—There is no inflammatory affection of which the diagnosis has been considered more difficult than pericarditis. Laennec states that he has often, on dissection, discovered the disease in a severe form, when nothing had afforded a suspicion of its existence; and, on the other hand, that he has frequently witnessed all its signs, without finding a vestige of the malady. Dr. Latham mentions two cases of what appeared to be, and was treated as, marked inflammation of the brain; yet this organ was found perfectly sound, and the heart affected with intense pericarditis.* Andral relates a similar case.† It is proper to keep these difficulties prominently in view, in order that practitioners may be better prepared to contend with them. But it must be added that such cases as those of Latham and Andral are extremely rare; and that, with the improvements in diagnosis introduced by modern research, the disease may, I feel assured from numerous post-mortem examinations, be nearly always detected.

I shall first enumerate the symptoms, and then endeavour to point out the causes of their obscurity, the means of rendering them available, and the diagnosis from other inflammatory affections.

The symptoms are as follows: acute inflammatory fever; a pungent, burning, lancinating pain in the region of the heart, shooting to the left scapula, shoulder and upper arm, but rarely descending below the elbow or even quite to it. The pain is increased by full inspiration, by stretching the left side, and especially by pressure between the præcordial ribs, and forcing the epigastrium upwards underneath the left hypochondrium. When the inflammation is only subacute, the pain is more or less dull, and does not lancinate. The next symptoms are, inability of lying on the left side, and sometimes in any position but one, which is most commonly on the back; dry cough; hurried respiration; palpitation of the heart, the impulse of which is sometimes violent, bounding and regular, though its beats may, at the same time, be unequal in strength; at other times it is feeble, fluttering, and irregular; pulse always frequent, and generally, at the onset, full, hard, jerking and often with a thrill. Sometimes it maintains these characters throughout, but more commonly it becomes, after a few days, weaker than accords with the strength of the heart's action and, in the worst cases, small, feeble, intermittent, irregular and unequal. Occasionally it possesses the latter characters from the commencement; whenever they exist, they are accompanied by dyspnœa; a constrained position, deviation from which induces a feeling of suffocation; extreme anxiety; a peculiar drawn or contracted appearance of the features, occasionally with the sardonic grin; faintness, constant jactitation, insupportable distress and alarm, cold perspiration, and, finally, from obstruction of the circulation, by intumescence and lividity of the face and extremities.*

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* Lond. Med. Gaz. vol. iii. p. 209.

† Clinique Medical, vol. iii. p. 444.

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* I have seen extensive adema of the feet supervene during the last twelve hours of life.

One cause of the obscurity of the above symptoms would, at first sight, appear to consist in their diversified, incongruous, and variable nature. The pulse, for instance, displays, at one time or other, almost every kind of character; the disease, though the inflammation be equally intense, is sometimes very supportable,—at others, agonizing; in one case it terminates fatally in two or three days,—in another it lasts as many weeks.

Now, in reality, these diversities, while they do not render the symptoms less pathognomonic of the disease in general, as will presently be shown, are invaluable indications in another point of view—they denote the nature and progress of the anatomical changes of structure, and, in correspondence, the progress and exact state of the malady. For it is ascertained by experience that a difference in the quality and quantity of the effusion, imparts a totally different aspect to the symptoms. Thus, when, either from the effusion consisting principally of concrete lymph, or from the simultaneously secreted serum being absorbed, universal adhesion of the pericardium promptly takes place, preventing all further fluid effusion, the action of the heart maintains throughout much the same vigour and regularity as it manifested at the onset of the malady, and the pulse exhibits corresponding characters of strength, hardness and regularity. Under these circumstances also, the position is less constrained, and less pain is produced by an unfavourable one; in consequence, perhaps, of the heart being curbed by the adhesion, and thus prevented from impinging with the same degree of violence against the thoracic walls. Finally, as the force and rhythm of the heart's action, and consequently the circulation, are adequately maintained, the life of the patient will be prolonged probably for weeks even though the inflammation remain unsubdued, and he will sink at last apparently from mere exhaustion by the effects of protracted irritation.

But should there be a copious serous effusion, the heart's action is mechanically embarrassed by the compression that the fluid occasions,—a compression which is the more considerable because the pericardium, deprived of its distensibility by inflammation, is incapable of yielding as the fluid accumulates. Hence the organ, unable to transmit its contents, becomes congested; it flutters, intermits, beats feebly, irregularly, and unequally. The pulse has corresponding characters and is sometimes scarcely perceptible. Hence supervene faintness, dyspnœa, anxiety, coldness, lividity, a sense of suffocation on the slightest deviation from a certain position, with all the other symptoms indicative of an obstructed circulation. If this state be not expeditiously relieved by remedies, the patient dies in the space of a few days or even hours.

Should the fluid be copious from the first, this series of symptoms will make its appearance equally early; but, in general, two, three, or four days elapse before the accumulation becomes considerable; in which case the former series will exist during this period, and will then be suddenly replaced by the latter. In a few instances I have found the latter exist when the quantity of fluid was inconsiderable, but that of lymph, enormous. I conceive, therefore, that an enormous accumulation of lymph has the same effect as fluid in embarrassing the action of the heart. I have also found the worst class of symptoms occasioned by a less quantity of fluid in some cases than in others,—a difference which probably depends, in some cases, on diversities in the nervous irritability; but, in others, I suspect that it is connected with the simultaneous existence of carditis; for, when the affection has been thus complicated, I have known the feeble, fluttering action of the heart and all its concomitant train of unfavourable symptoms, occur, though the effusion within the pericardium was inconsiderable. The peculiar expression of the features is occasioned by the sympathy subsisting between the respiratory nerves of the face and those of the heart.

Such are the causes of the symptoms. It will now be apparent that their variability is calculated to enlighten, rather than to perplex the practitioner, and that, whatever aspect they assume, they would still be abundantly sufficient, did no other difficulties interfere, to render the disease one of easy diagnosis. But, unfortunately there *are* other difficulties. These consist partly in the absence or mildness of some of the most important symptoms.

When pain in the immediate situation of the heart, increased by pressure in the interspaces between the ribs or upwards under the left hypochond, is accompanied by increased action of the organ and fever, there can scarcely be a doubt of the existence of pericarditis. But sometimes, though rarely, pain is totally absent; in which case, the practitioner must carefully employ pressure as above directed; and if, notwithstanding, no pain is felt by the patient, he must carefully turn his attention to the remaining symptoms. Should the pulse be feeble, faultering, intermittent, unequal &c. without any apparent adequate cause, (and it is well known to practical men that such a pulse rarely if ever exists in ordinary cases without an obvious cause,) this sign,* especially if attended with the usually concomitant signs of an obstructed circulation, affords evidence of the strongest description.

But there may neither be pain, nor an unsteady pulse. In this case should the action of the heart be violent and of a remarkably bounding or jerking nature without any manifest cause,—especially organic disease of the organ; and should it be accompanied by a greater degree of fever and anxiety than can be accounted for by any other existing complaint; finally, should it be attended with the stethoscopic signs presently to be described, the physician will seldom be wrong in diagnosticating pericarditis. The presumption is still stronger if, when the symptoms supervene, the patient is affected with acute or subacute rheumatism,—an affection which, whether severe or mild, whether in its early or its latter stages, is, beyond comparison, the most frequent cause of pericarditis.

It was an opinion of Corvisart that the most acute cases were the most obscure, because, says he, "the attack is abrupt, the progress rapid, and the termination almost sudden." This obscurity was felt by that acute observer, because he was

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[*] On it alone I have seen M. Chomel found a successful diagnosis in the last stage of a typhus fever, when the symptoms were extremely complex.

not acquainted with any signs of the disease on which he could depend but the feeble, unsteady pulse, the anxiety, dyspnœa, lividity and other symptoms dependent on obstruction of the circulation,—symptoms which did not always show themselves early enough to afford him data for the diagnosis before the case was hopeless. At present, however, when we are in possession of so many signs, the same obscurity does not exist. I have seldom experienced, nor have I seen other physicians experience, much difficulty in recognising the acute pericarditis to which Corvisart refers. The most obscure cases are those mentioned by Latham and Andral, in which a fictitious inflammation of the brain or any other organ diverts the attention from the heart, and the delirium of the patient renders it impossible to obtain information from himself. Still, when apprised that such cases exist, I should think it perhaps not impossible to provide against them. If, for instance, it were the general practice (one which I invariably pursue myself) to place the hand on the præcordial region as well as on the pulse in every severe inflammatory or febrile affection, in the same way that we daily feel the abdomen in cases of fever, even though the patient make no complaint of it, we should seldom fail to find an inordinately increased impulse or some other anomaly in the action of the heart, which would lead us to make a regular and probably successful investigation for pericarditis. For there can be little doubt that the symptoms, in the cases alluded to, are in reality not absent, but merely masked by others of predominant severity.

The only remaining cause of obscurity is, inflammation of some of the thoracic viscera, particularly the pleura, the pain of which may be seated over the heart. Pleurisy may be detected by œgophony, extensive dulness on percussion, and diminished or absent respiratory murmur. Peripneumony may, in addition to its ordinary symptoms, be recognised by the crepitous rattle, and deficient respiratory murmur and resonance on percussion; finally, bronchitis may be known by the mucous, sibilous and sonorous rattles. Should none of these signs be present, the negative evidence thus obtained fixes the disease on the heart. Should they be present, the diagnosis of the pericarditis must be made by a general comparison and cautious consideration of all the symptoms. In this case, should the affection of the heart be overlooked, the error is not one of the most serious kind, as the treatment for pleuritis or peripneumony is well adapted for pericarditis.

In a disease the treatment of which requires so much decision and promptitude in the practitioner as pericarditis, it is necessary for him to be thoroughly conversant, not only with the symptoms of deterioration, but also of amelioration. To these, therefore, I shall advert.

If the worst symptoms decline, namely the feeble, fluttering, unsteady pulse, the feeling of faintness and suffocation, and the constrained position to which that feeling confines the patient, we may be tolerably sure that the fluid, on which these symptoms commonly depend, is decreasing by absorption. But, notwithstanding, should pain, violent impulse, fever, and anxiety continue, the inflammation is in progress, and is adding to the accumulation of lymph, if not also of fluid. Should, however, the pain, instead of being fixed and pungent, become a diffuse uneasiness; should the anxiety decrease, and the peculiar vehemence of the heart's action gradually degenerate into the beat of a merely accelerated circulation, the inflammation may be presumed to be on the decline; but it is not until all these symptoms have completely ceased, that it can safely be said to have terminated.

Still—lymph and adhesion of the pericardium may remain, rendering the reparation imperfect; and such we may consider to be the case so long as, with every advantage of perfect tranquillity and abstinence, the motions and sounds of the heart do not completely regain their natural standard. Even though this be the case, nothing is more common than a recurrence of palpitation and other symptoms of diseased heart when the patient resumes his accustomed avocations. It is not, therefore, until, very gradually returning to corporeal exercise, he finds himself, after an adequate trial, perfectly capable of his wonted exertions, that the cure can be pronounced complete.*

Physical signs.—*The impulse* of the heart is greatly increased:—not only heaving the thoracic walls vigorously, but being remarkable for its abrupt or jerking character: whence it often shakes the whole anterior chest. Some beats are generally stronger than others, even when the action is regular. The pulse or rather throb of the arteries, often perceptible over the whole body, is of a corresponding nature, each undulation of the blood shooting with instantaneous velocity under the finger, as if through a lax or imperfectly filled tube, and constituting what is called a bounding, or, more expressively, a jerking pulse,—the pulse that we feel during reaction after uterine or other excessive hemorrhage. Very frequently it is accompanied with a distinct thrill. Sometimes it is stronger and more voluminous, at others, smaller and weaker; yet, in the latter case, it still retains the same jerking character.

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* See an excellent paper by Dr. Latham, Lond. Med. Gaz. vol. iii. p. 213, to whom the profession in general and myself in particular, are greatly indebted for his researches on this subject.

When the action of the heart becomes feeble and faltering, the impulse of the organ of course sustains a corresponding change; but, notwithstanding, the jerk accompanies any isolated contraction that happens to be strong.

An impulse and pulse of the jerking description denote an inordinately abrupt, and as it were spasmodic, contraction of the heart, probably attributable to an increase of irritability resulting from the inflammation. It is this peculiarity in the beat, which distinguishes it from the beat of a merely accelerated circulation, a distinction perfectly familiar to practical men. The peculiarity subsists not only during the continuance of inflammation, but so long as the action of the heart remains quick after the inflammation has apparently subsided,—a period which generally occupies several weeks, and, if adhesion of the pericardium has taken place, frequently as many months. I have known it exceed half a year. In very protracted cases, it is probable that the irritability of the heart is kept up either by an occasional recurrence of inflammatory action, or by the unnatural circumstances in which the organ is placed by adhesion, or, finally, by a softened state of the muscular substance, the result of carditis.

The sounds.—The sound of the ventricular systole is not only unusually sonorous, but is accompanied with a bellows-murmur. This sign was first noticed by Dr. Latham, who pointed it out to me at St. Bartholomew's Hospital in 1826. Since that time, I have never found it absent *when the heart presented the increased, jerking impulse* above described. Dr. Latham restricts his observation to rheumatic pericarditis: to myself the phenomenon has appeared to exist equally in every form of the disease. When the action of the heart has been feeble and faultering, I have found the murmur absent; but when, in the same case, the action has, either previously or subsequently, been strong and jerking, the murmur has existed. The reason of this will be obvious from the explanation which will presently be offered. The murmur sometimes continues after the heart appears to have resumed its natural action and the patient to be well; but, so long as it remains, as remarked by Dr. Latham,[*] "his return to the habits and exertions of health will bring back palpitation and other symptoms, which bespeak the certainty of mischief still abiding in the heart."

Not the ventricular systole only, but occasionally, though by no means always, its diastole likewise, is attended with the bellows-murmur; and I have found this supersede and, as it were, annihilate the natural second sound more completely in pericarditis, than, I think, in any other affection of the heart. Sometimes, in short, it is a pure whizzing, equally prolonged as, and almost continued into, the first sound. (Case of Harrison.)

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[*] Lond. Med. Gaz. vol. iii. p. 214.

What is the proximate cause and what the mechanism of these preternatural murmurs? That of the ventricular systole, I am inclined to attribute, mainly at least, to the increased velocity with which the blood is propelled in consequence of the morbidly abrupt contraction of the heart: an explanation which appears to me to be rendered probable by the following considerations, already fully developed in the article on murmurs independant of organic disease: 1. By repeated abstractions of blood, in animals, at intervals of a day or two, I have produced at pleasure the rapid, throbbing, jerking, and thrilling action of the heart and arteries and, in strict concomitance with it, the bellows-murmur.

2. The murmur takes place in nervous palpitation, when the action of the heart and arteries is of the nature described.

3. The loudness of the murmur observes a very accurate ratio to the violence of the throbbing, and it subsides when the throbbing ceases, unless there

remain an organic lesion, presently to be described, which generates it on a different principle.

But though I attribute the murmur of the ventricular systole *mainly* to the cause described, I believe that it may, in some instances, originate partly in another cause: namely, constriction of the arterial orifices consequent on inflammation of the lining membrane. For, as this membrane is more liable to inflammation where it constitutes the valves, than elsewhere, it is consistent with analogy to suppose that, by its intumescence and loss of elasticity, the orifices will undergo the constriction alluded to.

The murmur accompanying the second sound, I am inclined to attribute perhaps entirely to the same constriction, affecting the auriculo-ventricular orifices. This I infer, because I have not found it produced in any appreciable degree by abrupt, jerking action of the heart in reaction from loss of blood, and in nervous palpitation; and because, when I have noticed it in pericarditis, I have invariably found it connected with a more or less thickened and opake state of the valves—a state which, though perhaps scarcely amounting in every instance to an obstruction when it was examined in the dead subject, gives ample reason to believe that it might have constituted one during the period of acute inflammation. (Case of May, &c.) Should this be found true, the bellows-murmur of the second sound renders the prognosis more gloomy; as it bespeaks a more extensive inflammation and the probability of subsequent valvular disease.

Percussion.—When the pericardium contains much fluid, the resonance of the præcordial region becomes dull over a greater extent than natural. (Vid. p. 6.) The impulse, also, it may be added, is undulatory, and not exactly coincident with the first sound, in consequence of the heart having to displace the fluid interposed between it and the thoracic walls, before it can impinge against the latter. (Vid. Hydropericardium.) M. Louis states that he has found a temporary effusion of fluid cause a prominence of the cardiac region. I do not happen to have noticed this, but I think it very probable, especially in young subjects in whom the cartilages are soft.

Signs and Diagnosis of Chronic Pericarditis.

General signs.—The signs of chronic pericarditis are much the same as those of acute, but in a very inferior degree. The fever is more that of hectic or marcor, with occasional exacerbation when perhaps the inflammation becomes subacute. The anxiety and restlessness, though sometimes great, are comparatively supportable. The position is less constrained and I have observed that the patient often prefers the sitting posture with the body inclined forwards. The circulation is less embarrassed, and the action of the heart, though often abrupt and jerking, is usually somewhat feeble, except during any temporary exacerbation of inflammatory action. The pulse, also, is sometimes not very unsteady though the pericardium be full of fluid; which I attribute to the elasticity of the membrane not being so far destroyed by the inflammation as to prevent it from gradually undergoing extention, and accommodating itself to its contents. Whence compression of the heart by the fluid is in some degree obviated. The patient, I have thought, more frequently complains of a load and fulness, "something which he cannot get down," in the scrobiculus cordis, in chronic, than in acute pericarditis.

Chronic pericarditis, especially if such from its commencement, is more obscure than acute. I have, in former years, seen it overlooked more than once. But these cases, when I now revert to them, appear to me to have presented sufficiently characteristic symptoms. The history affords the greatest light. If the patient, previously exempt from disease of the heart, has suddenly become affected with its symptoms, attended by marcor and some degree of fever, within a period seldom extending beyond a few months, and which he often dates from a blow or fall on the breast, a rheumatic fever, or an inflammation with pain in the præcordial region, chronic pericarditis may be strongly presumed; and if these symptoms coincide with the physical signs of fluid in the pericardium, the existence of the malady may be regarded as almost certain.

Physical signs.—The impulse and pulse have much the same general characters as in acute pericarditis, except that, as the heart's action is less vigorous, they are not so strong. The sounds will vary according to circumstances. When the action of the heart is jerking and not wholly devoid of force, the first sound will be attended with a murmur, which, however, is generally very slight. When there is inflammatory constriction of the orifices, a murmur will attend both sounds. Should the heart be dilated, as is frequently the case, the sounds will be increased; and should hypertrophy be conjoined with the dilatation, the impulse will sustain a corresponding augmentation of force. The signs of fluid in the pericardium are the same as in acute pericarditis; namely the extensive dulness on percussion, and the undulatory impulse.

SECTION III.

CAUSES, PROGNOSIS, AND TREATMENT OF PERICARDITIS.

Causes, 116. *Prognosis*, 116. *Treatment of* ACUTE, 117. *Importance of Promptitude*, 117. *Venesection and Leeching*, 117. *Local Bleeding, when preferable to general*, 118. *Cupping, when preferable*, 118. *Purgatives*, 118. *Advantage of active Measures at first*, 119. *Diluents, Tartrate of Antimony, Diet*, 119. *Antiphlogistic Treatment insufficient without Mercury*, 120. *Mode and Extent of its Exhibition*, 121. *Blisters*, 121. *Sedatives—Hyoscyamus and Digitalis*, 122. *Treatment during Convalescence*, 122. *Relapses most common after Rheumatism*, 122. *Causes—Relapses more tractable than primary Attacks*, 123. *Treatment*, 123. *Should not be too active*, 123. *Remedies*, 124. *Treatment of* CHRONIC *Pericarditis*, 125. *Counter Irritants—Blisters, Plasters, Setons, Issues, Diet*, 125.

Causes of Pericarditis.—The most frequent causes are, blows or excessive pressure on the præcordial region, inflammation propagated from the lungs or pleura, and, far above all, rheumatism. From this cause, children and young persons suffer much oftener than others. The remaining causes are, those of inflammation in general; viz. cold, febrile excitement, &c.

Prognosis.—This disease, supposed by Corvisart to be necessarily fatal, has, by subsequent experience, been proved curable,—and completely curable; but as the possibility of effecting a complete cure is limited to a very brief period, and as, unless it be complete, the patient almost inevitably dies sooner or later from the consequences, the disease must be regarded as one of the most formidable incident to the human race. Some of these consequences I shall describe in the next section under the head of adhesion of the pericardium.

Treatment of acute pericarditis.—The antiphlogistic treatment, in as energetic a form as circumstances will allow, should be employed with the utmost promptitude. The loss of a few hours at first, may be irretrievable, and hence hesitation and indecision may seal the fate of the patient. If the attack is recent and the patient's strength will admit, blood should, in the first place, be drawn freely and by a large incision, from the arm, so as to bring him to the verge of syncope. From five and twenty to forty leeches, according to the strength, should then be applied to the præcordial region so soon as the faintness from the venesection disappears and reaction commences,—which generally happens in the course of from ten minutes to an hour or two. Unless the pain be completely subdued by these measures, the leeching, and in some cases the general bleeding also, may be repeated two, three or more times, according to the strength, at intervals of from eight to twelve hours; or, what is a better rule, so soon as the pulse and action of the heart denote a recommencement of reaction.

It is not, however, in every case, that so active a treatment is required. I have seen a single prompt and abundant application of leeches or a cupping at once subdue every formidable symptom. When the patient, either from age, a feeble constitution, or the advanced state of the malady, cannot bear extensive depletion, local bleeding is, according to my observation, decidedly preferable to general: but it should be practised effectually, —by cupping to twenty ounces or more, or by the application of from twenty-five to thirty or forty leeches. When, from depletion having already been carried to a great extent, or from the advanced stage of the disease, it is not safe to draw much more blood, yet it appears expedient, from the persistence of pain, &c. to draw some, I have generally found that a smaller quantity drawn by cupping produced more effect than a larger by leeching. The cause of this probably is, that, by cupping, it is drawn more expeditiously.

While the bleeding is in progress other means should not be neglected. The intestinal canal, if at all confined, should immediately be evacuated by a purgative enema. Three drachms of senna leaves, an ounce of sulphate of soda infused in a pint of boiling water, and strained, answers the purpose. At the same time, five grains of calomel with five or ten of comp. extr. of colocynth, and two or three of extr. of hyoscyamus, should be given, and, in two hours, be followed by a senna draught.

The strength of the remedies employed must in each case be apportioned to the vigour of the patient's constitution, but the object is the same in all—expeditiously to prostrate the action of the heart, and for a time to keep it prostrate by preventing the re-establishment of reaction. If this object can be accomplished for the first twenty, thirty, or forty hours, the disease frequently does not rally, but remains perfectly under the control of remedies. I feel satisfied that a degree of activity in the first instance, which to some may appear excessive, is an ultimate source of economy to the strength of the patient; for the disease is subdued at once, and the protracted continuance of depletory measures, the most exhausting to the constitution, is rendered unnecessary.

In addition to the above measures, diluent, cooling drinks, as four scruples of supertartrate, or two of nitrate of potass in a quart of water and flavoured at pleasure, should be allowed in unlimited quantity, in order, by diluting the blood to render it less stimulant to the heart. Nauseating doses of tartrate of antimony, as one-sixth to one-eighth of a grain, every two hours, may be employed with advantage. The diet should consist wholly of the weakest slops, as barley water, gruel, weak tea, arrowroot, &c.

But the antiphlogistic treatment alone is not to be relied upon: rarely, if ever, does it, in a severe case, effect a complete cure. The practitioner sees all his resources gradually exhausted, while the disease proceeds with an even, uncontrolled tenor, to its fatal termination. Sometimes, indeed, all the other symptoms disappear, but the action of the heart remains stronger than natural: at other times the heart even regains its healthy action and the cure appears complete; yet, in both these cases, the palpitation, accompanied with symptoms of organic disease of the heart, recurs when the patient resumes his accustomed occupations. The reason of this is very intelligible. Unless the effused lymph, as well as the serum, be absorbed, it causes an adhesion of the pericardium, and thus constitutes a destructive disease. Now antiphlogistic measures can neither prevent the effusion of lymph, nor with any degree of certainty cause its absorption. Mercury *can* do this,—as is visibly displayed in iritis; mercury, therefore, is the sheet anchor of the practitioner. Dr. Latham is of opinion that its success is restricted to the condition of its producing salivation, and producing it rapidly. From many observations I am satisfied of the general truth of this remark, and would, therefore, give the remedy on this principle; but I have seen cases in which cures, not falsified after many months, were effected, though salivation was not produced. The mineral, however, was freely administered, and probably produced its specific effect though not in an apparent manner. From five to eight grains of calomel, or from ten to fifteen of blue pill, prevented from purging by a grain or a grain and a half of opium, three times a day, commencing after the first bleeding and a purgative, generally produce the effect with sufficient expedition. Inunction may be superadded or partially substituted, if mercury, taken internally, disagree. A manifest abatement of the symptoms generally takes place immediately on the effect of the remedy becoming apparent in the mouth—especially if a free salivation is established within the first thirty or forty hours. It should be maintained for a week or ten days or even longer unless the symptoms completely yield before the expiration of this period.

Should pain continue in the advanced stages of the malady, and after the period for applying leeches has passed, blisters may be resorted to, and repeated in quick succession, with great advantage. I have occasionally found a third or a fourth necessary before the pain has been completely removed. In the repetition of blisters, as well as of leeches, cupping, and venesection, and in the selection of one of these remedies in preference to another, much must necessarily be left to the judgment of the practitioner. It is only experience which can teach the exact adaptation of remedies to the circumstances. It must also be left to his discretion whether to give sedatives or not. When the restlessness and nervous irritability were great, I have seen much benefit derived from tinct. hyoscyami m xv ad xx with the same quantity of tinct. digitalis, in a draught three or four times a day. Sedative remedies, however, should not be given until the first severity of the inflammation has subsided; nor should they ever be allowed, by producing their poisonous effects, to confuse the symptoms, already sufficiently complex, in the latter stages.

During convalescence, it is sufficient to say that a very spare unstimulating diet and extreme tranquillity must be imperatively enjoined until the action of the heart has become perfectly and *permanently* natural.

An individual who has recently been affected with pericarditis is peculiarly liable to a recurrence of it: especially if it has resulted from rheumatism and if the reparation has been incomplete. In this case, should rheumatism return, it rarely fails to be accompanied with a renovation of the pericarditic symptoms.

This cannot be a subject of surprise; for it is consistent with general analogy that a part recently injured by inflammation, is more susceptible than a healthy tissue of inflammatory action—the reason of which probably is, that the vessels of newly organized adventitious structures are more tender and irritable than others. Secondary inflammation, however, has not the same energy and intensity as that of a healthy structure, it yields more promptly to curative measures, and is more completely within the powers of medicine. Hence a first attack of pericarditis is more dangerous than any subsequent one. It is comparatively rare for a patient to die from the direct effect of a recurrent attack; and, what is still more remarkable, he may sustain several without being left in a materially worse condition than after the first. In the subjoined cases instances will appear in which attacks occurred from time to time during a period of ten or fifteen years.

Much discretion, however, is requisite on the part of the practitioner to bring such recurrent attacks to a favourable termination, and the danger of doing too much, is perhaps greater than that of doing too little. He must, in particular, be cautious of bleeding too extensively with the object of reducing the excessive energy of the heart's action; for this energy, he must recollect, is a consequence, not of the inflammation only, but partly also of an organic affection of the organ left by the primary attack. Nor is there the same motive for a vigorous employment of mercury. For, the heart being already irreparably disorganized, it would be chimerical to entertain the expectation of effecting a *perfect* cure. The object, therefore, should be, simply, to prevent deterioration by combatting the inflammation as it presents itself.

For the accomplishment of this object, a moderate use of blood-letting and mercury suffices; and leeching or cupping on the præcordial region is more efficacious and less exhausting than venesection. Blisters are, in these cases, peculiarly beneficial, and they may be repeated in quick succession as often as they are required and can be borne. When there still remains a little lingering pain, which scarcely authorises vigorous measures, but cannot prudently be left, the most valuable and convenient remedy has appeared to me to be, a plaster composed of a scruple of tartrate of antimony, four scruples of the emplast. picis comp. and two scruples of wax to diminish the tenacity of the adhesion.

In these cases, also, where the sufferings of the patient, though perhaps not severe, are very protracted, and accompanied with much loss of rest, great advantage is derived from a pill of from three to six grains of extr. of hyoscyamus at bedtime, and moderate doses of tincture of digitalis during the day, the specific poisonous effect of the latter remedy being obviated by omitting it for a couple of days after every three or four.

Chronic pericarditis.—When pericarditis is essentially chronic, and the cavity appears to contain fluid, counter irritant remedies are the most suitable. After what has already been said, it will be sufficient merely to mention blisters, either in succession or kept open with savine cerate, the tartrate of antimony and pitch plaster, and likewise issues and setons. The last remedy, however, generally creates so much irritation as to do more injury by deteriorating the general health, than good, by its local effect. Mercury to a moderate extent, may, if discreetly employed, be advantageous by promoting absorption; but, in general, the patient is too much reduced by constitutional irritation, to admit of more than the mildest action of this remedy.

The diet may, in chronic cases, be more nutritious comprising light animal food and broths.

SECTION IV.

ADHESION OF THE PERICARDIUM.*

Adhesion frequent, especially from Rheumatism, 126. *Incompatible with Health, according to the older Authors*, 126. *Laennec and Bertin of the opposite Opinion*, 126. *The latter probably wrong*, 127. *Its effects, according to the Writer's Observation*, 127. *How it occasions Hypertrophy—how Dilatation*, 128. *Its pathognomonic Signs*, 129. *Retraction of the Epigastrium not to be depended upon*, 129. *Prominence of the Cardiac Region and high Pulsation*, 129. *Jogging Motion*, 130. *Bellows-murmur*, 131.

PERICARDITIS, both acute and chronic, and especially that originating in rheumatism, frequently terminates in adhesion of the pericardium. Lancisi, Vieussens, Meckel, Senac, and Corvisart, are of opinion that, with a complete and intimate adhesion, the patient cannot live in a state of health. I know not how it is that Laennec and Bertin have formed an opposite opinion. The former states that he had opened a great number of subjects so affected, who had never *complained* of any derangement in the circulation or respiration; whence he infers that adhesion often does not in any respect interfere with the exercise of those functions.*

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* This should, strictly perhaps, be ranged amongst the organic affections; but, as it is in some cases more or less inflammatory, and as it is intimately connected with pericarditis, it cannot be separated from it without breaking the continuity of the subject.

My own experience is entirely opposed to this doctrine. The *complaints* of the patient are, perhaps, not a just criterion; for I have often found the working classes disclaim dyspnœa when labouring under enormous hypertrophy and dilatation and when that symptom obviously existed in a great degree. Many others also, especially children, are naturally inattentive to their own sensations, and close interrogation is the only mode of ascertaining that, after the attack of pericarditis, they became incapable of some exercises, habits, or efforts which they previously accomplished with facility.

I have never examined a case of complete adhesion of the pericardium without finding enlargement of the heart,—generally hypertrophy with dilatation. I have observed that cases of adhesion terminating in enlargement, often hurry to their fatal conclusion with more rapidity than almost any other organic affection of the heart; and I have, on the other hand, repeatedly seen patients die from the consequences of an adhesion, the history of which I could trace back eight, ten, or more years; yet such individuals would not unfrequently represent their health to have been perfect during the greater part of that period, and would not admit, until closely interrogated, that they had been more or less "short-winded." Hence I infer that, though adhesion may not, for a time, create much inconvenience, its effects are ultimately fatal.* It appears to me that a tranquil, abstemious life, by which, in other forms of organic diseases of the heart, existence may sometimes be prolonged to its natural period, cannot be equally availing here; for, as the action of the organ itself is a constant struggle, repose is impossible.

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* De l'Auscult. tom. ii. p. 664.

How adhesion occasions hypertrophy is easily understood; for the organ must increase its contractile energy, in order to contend against the obstacle which the adhesion, by shackling its movements, presents to the due discharge of its function; and, as explained in the article on hypertrophy, increased action leads to increase of nutrition. The cause of the co-existent dilatation is not less manifest. As the shackled organ transmits its contents with difficulty, it is constantly in a state of greater congestion than natural, and, as is more fully explained in the article on dilatation, permanent distention is the most effective cause of this affection. When the muscular substance is softened, as frequently happens, dilatation takes place much more readily in consequence of the deficient elasticity or tone of the heart's parietes.

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* This refers, of course, to intimate, not to loose adhesion.

When adhesion of the pericardium has produced hypertrophy with dilatation, its history identifies itself with that of the latter maladies, of which it renders the symptoms more severe and the progress more rapid. To avoid repetition, therefore, I refer the reader to the article on hypertrophy, and shall, here, only describe the signs which are pathognomonic of adhesion.

These signs have generally been considered very obscure. Dr. Sanders believed that he had discovered one of a positive nature in a dimple or retraction taking place, as he states, during the ventricular systole, in the epigastrium immediately below the left false ribs. I have searched for this attentively in several cases of adhesion, but have not been able to detect it in any degree which could constitute a sign. Laennec, who was equally unsuccessful, thinks that it could not take place unless the stomach, by adhering both to the diaphragm and the abdominal parietes, formed the medium of retraction.

In five or six cases I have remarked one sign, which has not, to my knowledge, been hitherto noticed: namely, the heart, though enlarged, beats as high in the chest as natural and sometimes occasions a prominence of the cartilages of the left præcordial ribs. (Cases of May, Harrison, a Boy, Payne, &c.) We should, indeed, naturally expect that the adhesion would brace up the organ, and that, when enlarged and not able to descend, it must, being bounded behind by the spine, force the walls of the præcordial region forward.

Another sign, and perhaps the most characteristic of all, is, an abrupt, jogging or tumbling motion of the heart, very perceptible in the præcordial region with the cylinder. It is more distinct when the heart is hypertrophous and dilated; and, under these circumstances, I have found the jogs correspond with the ventricular systole and diastole respectively, that of the diastole being sometimes nearly as strong as the other, and having the character of a receding motion. (Cases of May, Payne, Harrison, a Boy.) This jogging motion is distinguished from the undulatory movement of fluid in the pericardium, both by its nature, by the synchronism of the jogs with the sounds, and by the feeling that the heart, at each systole, comes in immediate contact with the thoracic walls.

A third sign consists in a bellows-murmur with the first sound, which I have always found present when the heart is enlarged and acting vigorously. Nor is it, in every case, confined to the heart: I have often heard it in the aorta, and formerly experienced difficulty in discriminating it from the murmur of dilatation of this vessel.* (Cases of Payne, May.) Although, when the heart is dilated, the murmur in question may be occasioned partly by the relative smallness of the orifices, and the greater angles at which the currents meet in them in consequence of the unusually rounded form of the ventricles, as already explained, p. 63. it is also, I believe, occasioned in a great measure by the sudden velocity with which the fluid is propelled, as it would not otherwise exist in the aorta.

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* Vid. Aneurism of the Aorta.

CHAPTER II.

CARDITIS, OR INFLAMMATION OF THE MUSCULAR SUBSTANCE.

1. UNIVERSAL. *One Instance only with general Effusion of Pus,* 132. *Laennec's Disbelief in the Existence of universal Carditis, ill-founded,* 133. *Other Instances, in Softening and Induration with Change of Colour,* 133. *Doubts of Laennec whether they are Inflammatory,* 133. *Cases in which these Characters penetrate only a certain Depth from the Surface,* 133. *Treatment the same as for Pericarditis,* 134. 2. PARTIAL. *Common,* 134. *Ulcers, Abscesses,* 134. *Their Signs,* 135. *Ulceration the most frequent Cause of* Rupture of the Heart, 135. *Rupture from other Causes—as Softening,* 135. *Cause of its generally occurring in the left Ventricle, explained,* 135. *Rupture of the Columnæ Carneæ and Chordæ Tendineæ —Symptoms,* 136. *Rupture of the Heart or great Vessels into the Pericardium, why not always immediately Fatal,* 136. Gangrene, *its Occurrence probably impossible,* 137.

INFLAMMATION of the muscular substance of the heart may be 1. universal, 2. partial.

1. Of universal carditis with *effusion of pus generally* throughout the muscular tissue, there is not, to my knowledge, more than a single instance on record, and that occurred to Dr. Latham. "The whole heart," says he, "was deeply tinged with

dark coloured blood, and its substance softened; and here and there, upon the section of both ventricles, innumerable small points of pus oozed from among the muscular fibres. This was the result of a most rapid and acute inflammation, in which death took place after an illness of only two days."[*] Laennec, never having met with, or heard of, a case of this kind, and considering an effusion of pus the only unquestionable sign of carditis, says, "there does not perhaps exist a single incontestible and well described example of *general* inflammation of the heart either acute or chronic."[†] Independent of the above instance, however, there are probably many others, which, though not attended with effusion of pus, will come under the denomination of universal carditis. For few will concur with this distinguished writer in excluding from the proofs of carditis, softening and induration with increased, or diminished colour of the organ. These are results of inflammation in other muscles, and analogy points out that they have the same origin in the heart. Further evidence is derived from the fact that, in cases of pericarditis, the characters in question sometimes occupy only a certain depth of the exterior surface of the organ; whence the presumption is almost positive that they originate in an extension of the inflammation from the pericardium. The cases of this description that are on record, are too numerous to be quoted. Several have fallen under my own observation. In this point of view, then, general carditis is not very rare.

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[*] Lond. Med. Gaz. vol. iii. p. 118.

[†] De l'Auscult. ii. p. 554.

As softening and induration are of sufficient importance to demand separate articles, I refer the reader to them for all that remains to be said on general carditis. They are introduced amongst the organic, rather than the inflammatory affections, because authors are not entirely agreed whether they result from inflammation or from other causes.

I have already stated, when treating of pericarditis, that this affection is greatly aggravated by the coexistence of carditis. As the treatment of the two is the same, it is unnecessary here to enlarge on it.

2. Partial carditis, characterized by the existence of an abscess or ulceration in the walls of the heart, is not very uncommon. Bonetus, in his Sepulchretum, has described a considerable number of cases. Abscesses are more rare than ulcers. The latter occur both on the external and the internal surface of the heart, and are consequent on inflammation of the membranes of those surfaces. The external ulcer is uncommon, but Oläus Borri-

chius, Peyer, and Graetz have left perfect descriptions of it. The first says " Cordis exterior caro, profundè exesa, in lacinias et villos carneos putrescentes abierat." The internal ulcer is more common. Bonetus, Morgagni, and Senac present many cases. I have met with two or three.

An ulcer, whether external or internal, may perforate the heart.

The signs of abscesses and ulcers vary in different subjects, and are not distinguishable from those of other affections. " I know not," says Laennec, " if auscultation will afford any more sure signs, and I avow that I think not."

Ulceration is the most frequent cause of rupture of the heart,—fortunately a very rare occurrence. Rupture independent of ulceration generally originates in some disease of the muscular tissue, by which its cohesion and resisting power are diminished :—softening for instance. I have met with one instance, mentioned in the article on dilatation. Haller and Morgagni describe many. It is generally in the left ventricle that the rupture takes place, a circumstance which at first appears remarkable, since this ventricle is the stronger; but, for the same reason, it contracts more energetically, and, as the rupture occurs during the contraction, we have thus an explanation of the phenomenon. It might be objected that supposing the strength of the muscle and the energy of its contraction to be in the direct ratio of each other, the explanation offered would not account for the phenomenon. To this it may be replied, that it is only strong muscles which *do* undergo rupture from the energy of their own contraction. Hence rupture of the auricles is much more rare than that of the ventricles.

The exciting causes of rupture are generally, considerable efforts, paroxysms of passion, external violence—as falls, &c.

Corvisart was the first who noticed and described cases of rupture of the fleshy columns and tendinous cords of the heart: Laennec and Bertin have each met with an instance of the same. Violent efforts, as coughing, were the cause; the symptoms were, sudden and very severe suffocating dyspnœa, followed by all the general phenomena of disease of the heart.

Rupture of the heart or great vessels into the pericardium is not always immediately fatal, as a solid coagulum or a fibrinous concretion has in several instances been known to arrest the hæmorrhage for a few hours.* Of ten cases mentioned by M. Bayle eight died instantaneously, one in about two hours, and another in fourteen.†

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* Case by Cullerier. Journal de Med. par M. M. Corvisart, Serone et Boyer, Sept. 1806, t. xii. p. 168.

† Revue Med.

The existence of gangrene of the heart has never been distinctly proved, and the following reasons lead to the belief that its occurrence is perhaps impossible; first, the muscular tissue is one of those least susceptible of it; and secondly, inflammation of the heart sufficiently intense to occasion it, is fatal to the patient before gangrene can take place. The cases on record of reputed gangrene, appear to have been nothing more than softening, which incipient putrefaction had rendered more analogous to gangrene.

CHAPTER III.

INFLAMMATION OF THE INTERIOR OF THE HEART AND ARTERIES, AND THE ALTERATIONS OF STRUCTURE CONSEQUENT ON IT.

Much difference of opinion has subsisted amongst authors respecting inflammation as occurring in the interior of the heart and arteries. Some, amongst whom Laennec holds the most distinguished place, have thought it extremely rare; others have attributed to it nearly all the morbid appearances and changes that are found in those parts. It will be convenient to consider the subject in separate sections, under the heads of acute, and of chronic arteritis, ranging under each its proper anatomical characters, and the morbid alterations that appear to result from, or appertain to it. The symptoms and treatment, will be reserved for subsequent sections.

SECTION I.

ANATOMICAL CHARACTERS OF ACUTE ARTERITIS.

ANATOMICAL CHARACTERS. A. Redness of the internal Membrane of the Heart and Arteries. *Sometimes Inflammatory and sometimes not*, 140. *Description of Redness* *not Inflammatory*, 140. *Of Scarlet Redness, in Aorta, Cavities of Heart, Valves, Pulmonary Artery*, 141. *Free from thickening or vascularity*, 143. *Of brownish or violet Redness*, 143. *It penetrates more deeply*, 143. *Redness alone not a Proof of Inflammation*, 143. *In what Subjects the Scarlet Redness occurs*, 144; *in what, the Brown*, 144. *Both are accompanied with softening of the Heart*, 145. *Symptoms of Scarlet Redness obscure*, 145. *Proof that the Redness is from Imbibition,—from Cases*, 145; *from Experiment*, 146. *Conclusions*, 147. *Description of inflammatory Redness*, 147.

B. Effusion of Lymph on the Internal Membrane, with thickening, &c. 148. *Arteries thus obliterated*, 149. *Lymph in the Aorta, Auricles, and on the Valves*, 149. *Lymph the only certain Sign of Inflammation, according to Laennec*, 150. *Fallacies to which other Anatomical Signs are liable*, 150. *Lymph from Chronic Inflammation*, 151. *Aorta obliterated by it*, 151; *also Arteries near Vomicæ, Tumors, &c.* 151.

C. Ulceration of the Internal Membrane—*from Inflammation*, 151. *From previous Chronic Disease, as Bone, Atheroma, &c.* 152. *These Ulcers described*, 152. *Originally Solutions of continuity*, 153. *The most frequent Cause of consecutive false Aneurisms*, 153. *Pustules*, 153; *originate under the Internal Membrane*, 153. *Ulceration within the Heart, rare*, 154. *Arteritis never terminates in gangrene*, 154; *but Arteries slough with contiguous parts*, 154. *Erysipelatous Arteritis from Injuries—very dangerous*, 155.

THE anatomical characters of acute arteritis are, redness of the internal membrane of the heart and arteries, an effusion of plastic, pseudo-membranous lymph on its surface, and thickening and ulceration of its substance. Each of these characters will be considered in succession.

A. *Redness of the internal membrane of the heart and arteries.*—This is sometimes inflammatory, and sometimes not. The redness not inflammatory, often appears in the aorta, the pulmonary artery, and the heart, and is a uniform intense colour, as if stained by the blood. Corvisart avows that he cannot give a satisfactory account of its nature and cause.* P. Frank regarded it as an inflammation of the arteries, which, according to him, occasions a peculiar and almost always fatal fever.† Bertin and Bouillaud have considered it, whatever was its shade, as the result of inflammation.‡ Laennec entertains an opposite opinion, and demonstrates satisfactorily that the redness in question, when not accompanied by other anatomical characters of inflammation, is the result of sanguineous imbibition. As it is necessary that the reader be able to judge for himself, I shall give some account of this redness, adhering to the description of Laennec; which I have verified by repeated experiments and dissections.

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* Corvisart. p. 36.

† De Curand. Homin. Morbis. tom. ii. p. 173.

‡ Traité, &c. p. 55.

The redness is sometimes scarlet, and sometimes brown or violet. The scarlet redness of the interior of the arteries is often confined to the internal membrane exclusively; and, when that membrane is removed by scraping with the scalpel, the fibrous coat is found underneath as pale as in its natural state. But in other cases the redness penetrates more or less deeply into the fibrous coat, and sometimes it reaches in parts even the cellular or external tunic. The redness of the internal coat is a perfectly uniform tint, similar to that which would be presented by a piece of parchment painted red. No trace of injected capillaries can be distinguished in it; but the tint is sometimes deeper in one part than another. Sometimes it diminishes insensibly from the origin of the aortà to the place where the redness ceases: but, very often, it terminates suddenly and by forming abrupt borders of an irregular shape. Sometimes, in the midst of an intensely red portion, is found an accurately circumscribed patch of white, which produces precisely the effect that is occasioned by an impression of the finger on a part of the skin affected with phlegmon or erysipelas. When the aorta contains very little blood, the redness only exists in the tract in contact with it, and forms a sort of ribbon. The origin and arch of the aorta are the parts of that artery which are the most frequently found thus reddened. Sometimes nearly all the arteries present the stain. The aortic and mitral valves then participate in it, and appear as if they had been plunged in a red dye. Though the red is scarlet in the arteries, it is deeper on the valves, approximating slightly to purple or violet.

When the pulmonary artery is reddened, its valves and the tricuspid are also very commonly stained in the same way. The stain of the right cavities and vessels of the heart is always of a deeper and browner hue than that of the left—a circumstance dependent, in all probability, on the darker colour of the venous blood. The internal membrane of the ventricles and auricles sometimes does not present any sensible change of colour, even when the valves are the most deeply reddened. Still, it is not rare for the internal membrane of the auricles to participate in the redness, and in this case, its tint resembles that of the valves. More rarely, the interior of the ventricles also presents a similar redness, but ordinarily it is browner or more violet. Sometimes the internal surfaces of the ventricles and auricles are the only parts reddened; but this occurs only when the heart is gorged with blood and the arteries contain scarcely any.

The redness above described is not accompanied with any sensible thickening or vascular injection of the stained membranes. A few hours' maceration in water suffices to make it totally disappear.

Such are the characters of the scarlet redness. We next come to the brownish or violet stain. It is found equally in the aorta, the pulmonary artery, the valves, the auricles and the ventricles. Most commonly, indeed, it is observed in all these parts simultaneously. It is often very unequal in intensity, and is always more marked on the parts of the vessels which, according to the laws of gravity, have been most in contact with the blood. It is not so commonly restricted to the lining membrane as the scarlet redness; for the muscular substance of the auricles and ventricles, and even the fibrous tunic of the aorta and pulmonary artery, participate in the dye—at least, in some points and to a certain depth.

Such is Laennec's account of redness of the internal membrane. But redness, he contends with great justice, is not sufficient to characterize inflammation, particularly when it is not accompanied by thickening or vascular injection of the reddened parts. Moreover, the exact circumscription of the redness in some cases, and its abrupt termination in others by geometrical, though irregular lines, (an appearance never seen in inflammation of serous membranes, though it presents itself occasionally and to a slight degree in that of mucous*) banish the idea of inflammation, and convey that of a stain by a coloured liquid which had run irregularly on the reddened membrane, and which, on account of its deficient quantity, had not been able to touch every part. Again, the circumstances under which the redness is usually found, countenance the idea of its being a stain rather than from inflammation.

Laennec has found the scarlet red to occur after a somewhat protracted agony in subjects still vigorous, but cachectic in consequence of disease of the heart or otherwise. The blood in these cases was never very firmly coagulated, and the body most frequently presented some signs of decomposition.

The brownish or violet red, he found in those subjects especially, who had died of continued typhoid fevers, of emphysema of the lungs, or of diseases of the heart. Almost all had experienced a long and suffocative agony; in all, the blood was very liquid and evidently altered, and signs of premature decomposition existed in the bodies. I have myself also very constantly found it in cachectic subjects affected with passive hæmorrhage from the gums, from ulcers, or from any tender or broken surfaces,—as in scurvy. It is, moreover, in summer particularly, and in subjects that are opened more than twenty-four hours after death, that the dark discoloration is most frequently met with.

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* I have seen it in the trachœa in variola. See also Bretonneau on Diptherite.

Both varieties of redness, and particularly the brownish or violet are accompanied with a greater or less degree of softening of the heart, and with an increased humidity of the arterial walls. In most instances these states are evidently the effects of a commencement of putrefaction.

Laennec has strong doubts whether the scarlet redness ever produces symptoms sufficiently severe and constant to render it capable of being recognized. He has found it in subjects who had died of widely different complaints, and he was never able to predict it by any constant sign.

The cases which Bertin and Bouillaud have adduced in substantiation of their opinion that the redness in question is of an inflammatory nature, are strikingly corroborative of the opposite views of Laennec. For, of twenty-four cases, eleven are typhoid fever, or other affections, in which there was a manifest alteration of the liquids and premature putrefaction. The thirteen other cases consist almost entirely of consumptive patients; and the writers observe, in general terms, that the redness appeared to coincide with a remarkably fluid state of the blood. It must, further, be remarked that most of their examinations were made in summer, and more than thirty hours after death.

In order to ascertain experimentally whether blood could occasion a stain, Laennec enclosed a quantity in a sound and recent aorta, and placed the preparation in the stomach of the subjects, in order to preserve it from drying and put it under the same circumstances of decomposition as the rest of the body. In twenty-four hours it presented a perfect specimen of the scarlet dye, which was not weakened by reiterated washing.

He found that blood too firmly coagulated causes imbibition feebly and slowly: that blood half coagulated, and particularly the blood, still slightly florid, which may be pressed out of the lungs, produces the scarlet redness: that very liquid blood, and particularly that with a serous intermixture, produces a violet colour of greater or less depth: and that if the artery be only partly filled, the dye occupies those parts alone which are in contact with the blood, thus forming a ribbon. If the walls of the artery are firm and elastic, the dye, he continues, requires a long time (seventy or eighty hours) for its formation, and is never very deep; but if, on the contrary, the walls are soft, supple, and charged with humidity, the dye promptly penetrates through the whole thickness. Warm weather and the rapid progress of putrefaction are favourable to the imbibition.

Boerhaave and Morgagni also attributed the red colour to the stagnation of blood which takes place during the agony in diseases accompanied with great oppression; and Hodgson likewise maintains that arterial redness, such as that above described, does not arise from acute inflammation, as it is not accompanied by any other anatomical characters of inflammation. When occurring in the vicinity of coagula, it is, he thinks, an effect of imbibition after death.

It is impossible not to conclude from all the evidence now adduced, first, that redness of the internal membranes of the heart and arteries cannot *alone* prove inflammation, secondly, that it is a phenomenon taking place during the agony, or after death, whenever it is found in conjunction with the following circumstances: namely, a prolonged and suffocative agony; manifest alteration of the blood; and a somewhat advanced decomposition of the body.

Such is the redness of the internal membrane of the heart and arteries which is not inflammatory. We now proceed to that which is. The colour of the latter may be the same; for the membrane, though inflamed, is still liable to imbibition. In the absence of imbibition, the redness is fainter, less shining, more equably diffused, and less characterized by streaks, patches, isolated unstained spots, and abrupt edges.

B. *Effusion of lymph on the internal membrane, with thickening of its substance.*—Whether redness be due to vascularity alone, or to this conjoined with imbibition, its inflammatory nature is known by the presence of other anatomical characters of inflammation. These are, thickening, swelling, and puffiness of the inner membrane; an effusion of lymph on either its free or adherent surface; and a preternatural vascularity, with softening and thickening, of the middle arterial coat. Each of the coats, also, may be separated from the other with much greater facility than natural, by scraping with the nail or scalpel. The internal and middle coats, in short, present all the phenomena of the adhesive inflammation as it displays itself in other membranes. It is by this inflammation that, if an artery be wounded or divided; if it be compressed by a ligature or tumor; or if it be simply irritated by ulceration of the surrounding parts or a pulmonary vomica; an effusion of lymph takes place into the cavity of the vessel and into the cellular tissue both investing it externally and connecting its several coats together, by which the caliber of the vessel is obliterated and hæmorrhage prevented.

Although obliteration never takes place in the aorta from inflammation alone, this vessel sometimes exhibits the vestiges of acute inflammation more palpably than smaller arteries. Thus, in a case seen by Dr. Farre the internal coat of the aorta was of a deep red colour, and a considerable effusion of lymph had taken place in its cavity. The lymph was very intimately connected with the internal coat.* Similar instances have been seen by Hodgson,† Bertin,‡ and apparently by Portal,§ and Morgagni and Boerhaave.‖ Lymph has been found effused within the auricles and on the valves by Baillie,¶ Laennec,** and Burns.†† I have met with it both in the heart and aorta. Effusions of lymph within the heart and great arteries, however, are very seldom *found;* and hence it is, that Laennec thinks inflammation of the internal membrane of those parts extremely rare.* But the presence or absence of lymph is not sufficient to determine whether inflammation exists or not; for, in many instances, the lymph, when first effused, is unquestionably washed away by the force of a circulation so powerful as that in the heart and aorta.

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* Hodgson on the Arteries, p. 5. and plate I. fig. 5.

† Ibid. p. 6.

‡ Case ii. p. 7.

§ Cours d'Anatomie Médicale, tom. iii. p. 127.

‖ Morgagni Epist. xxvi. art. 36.

¶ Morbid Anat. Edit. 5. p. 85.

** De l'Auscult. tom. ii. p. 127.

†† On Diseases of the Heart, chap. 9.

Laennec considers a layer of lymph on the internal membrane of the heart or great vessels to be the only incontestible sign of inflammation of that membrane, and, with ulceration, the only certain one:† for he doubts whether redness of the membrane, even when conjoined with thickening, swelling, puffiness, and an unusual developement of small vessels in the middle tunic, would satisfactorily prove inflammation in a subject considerably infiltrated, and whose tissues were very humid.‡ This is a point which can only be determined by the judgment of the physician in individual cases. I recently met with a case precisely such as Laennec describes, and, at the first glance, it was supposed by myself, and two eminent medical friends to be inflammatory, but a brief inspection proved the reverse. It is necessary, however, to be alive to this source of fallacy.

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* De l'Auscult. ii. p. 498.

† Ibid. p. 607.

‡ Ibid.

Kreysig, Hodgson, and Bertin and Bouillaud are of opinion that lymph effused by inflammation is the source of fungous or warty vegetations of the valves. Laennec rejects this opinion and attributes the vegetations to sanguineous concretions which adhere to the internal membrane and become organized. He does not deny, however, that an inflammatory false membrane may become the nucleus of these concretions. This subject will be more fully considered under the head of Vegetations.

Effusions of lymph are, in some instances, the result of chronic inflammation; being found in the vicinity of ossifications of arteries and other morbid alterations of a chronic nature. I have seen an ossified common iliac obliterated by a plug of lymph. Hodgson has seen nearly the same; and Dr. Goodison found the aorta itself obliterated by lymph, at a part where its whole circumference was converted into a bony cylinder. This lymph must have been secreted gradually; as the force of the aortic circulation would have washed away so copious an effusion, if soft and recent. It is probable that the obliteration of arteries in the neighbourhood of vomicæ and other ulcerations, and of tumors, is also occasioned by chronic inflammation.

C. *Ulceration of the internal membrane.*—Ulceration of the internal membrane sometimes takes place from acute inflammation, and it may exist without occasioning any lesion of the subjacent tissues. This, however, is rare; for, in general, the ulceration is a consequence of some previous chronic degeneration of the coats of the vessel, and is, in the first instance, rather a solution of continuity than an ulceration. Such is the case when it is occasioned by the detachment of calcareous incrustations, or by the deposition of atheromatous or other matter underneath the internal membrane. Ulcers from these causes are not uncommon, and though they do not, strictly, come under the head of acute arteritis, they may be conveniently considered in this place.

They vary in size from that of a mustard seed to that of a pea or bean, have more or less thick and ragged edges, and are sometimes so deep as to reach, and even to perforate, the external or cellular tunic. Laennec describes the formation of these ulcers from calcareous incrustations in the following manner. "When a calcareous incrustation is detached from the aorta, the species of sinus left by it is filled up by fibrine, which becomes, by decomposition, of the consistence of friable paste, and is often intermixed with phosphate of lime." This paste, when soft and pulpy, has been denominated *melicere*, or *atheroma*. Not unfrequently, the borders of the lesion are reddened for a little distance; and this he attributes to imbibition of blood, (rendered more easy in an altered structure,) rather than to chronic inflammation, which he thinks is not proved either by the presence of pus, or of any symptoms, local or general, that can be referred to it. These lesions, therefore, he regards as being, in the first instance, merely solutions of continuity from an entirely mechanical cause, and not ulcers occasioned by inflammation. He does not deny, however, that the oldest and most extensive of them sometimes become ulcers; for the internal membrane of the borders of the lesions is slightly tumid and red, and the surface of the fibrous tunic at their base is manifestly altered. But he contends that the inflammatory action which gives them the character of ulcers, is the effect, not the cause, of the solution of continuity. Solutions of continuity occasioned by the detachment of calcareous incrustations, are among the most frequent causes of consecutive false aneurisms.

Small pustules filled with pus, sometimes, though rarely, present themselves under the internal membrane of the aorta, and burst into its cavity. It is probable that they form the genuine or primitive ulcers of that vessel—those which are the most frequent cause of its perforation. They sometimes throw out curdy, and even calcareous matter. Laennec thinks that these pustules are occasioned by inflammation not of the internal, but of the middle arterial tunic, or of the fine cellular tissue which unites the middle to the internal tunic; and he is of this opinion, because, in inflammation of all membranes, as the peritoneum, the pleura, the mucous membrane lining the bronchia, pus forms on their free, and not on their adherent surface. Hence, as this secretion is found on the adherent surface of the inner arterial membrane, he infers that the inflammation occasioning the pustule, is seated, not in that membrane, but in the subjacent tissues. Pus is scarcely ever found on ulcers of the heart and arteries, because it is washed away as soon as secreted. For the same reason, it is never found on the internal surface of arteries that are inflamed without being ulcerated, though analogy leads us to believe that, under these circumstances, it is secreted.

Ulcerations are extremely rare within the cavities of the heart. I recently met with an instance, originating in a curdy deposition under the lining membrane.

I have never seen or heard of a case in which inflammation, when confined to the interior of an artery, terminated in gangrene. Arteries, however, are frequently involved in the sloughing of surrounding parts; in which case the blood generally coagulates in the vessels to a considerable extent above the line of sphacelation, and thus prevents hæmorrhage.*

It remains to be noticed that arteritis is sometimes of an erysipelatous nature. I allude to that which follows an injury of an artery, as by a ligature, a gun-shot wound, &c. especially if there be deep-seated disease in the muscles of the part affected. The inflammation, in these cases, sometimes runs along the internal coat of the artery till it reaches the heart. It is a most formidable disease, rapidly producing great irritative fever, an extremely quick pulse, complete collapse, low delirium, and generally death.

SECTION II.

ANATOMICAL CHARACTERS OF CHRONIC ARTERITIS, AND DISEASES OF THE COATS OF ARTERIES CONNECTED WITH IT.

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* Hodgson, p. 17.

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ARTERIES are more subject to chronic, than to acute inflammation. The internal membrane, when affected with it, is thickened, softened, and of a deep dirty red colour. These appearances are not uniformly diffused, but are more marked in the vicinity of calcareous and other degenerations. Hence some have supposed that these degenerations were the cause of the inflammation. There can be little doubt that they tend in many instances to keep it up; but it is highly probable that the degenerations themselves were originally caused by increased vascular action of a chronic nature. Whether the increased vascular action be in every case, particularly in those of calcareous depositions, strictly inflammatory, we shall inquire after bringing the various degenerations under review. Meanwhile it may be remarked that the appearances in arteries presented by chronic inflammation accompanied with morbid depositions, have been well known to authors from a very early period. The ancient physicians ascribed them to acrimonious, syphilitic, and scorbutic humors pervading the system. Some modern writers also, particularly Corvisart, Scarpa, Richerand and Hodgson, impute them to similar causes, especially to the syphilitic virus, or the mercury used for its eradication.

Morbid alterations in the coats of arteries, and especially the aorta.—The morbid alterations in the interior of the aorta which appear to be of chronic formation, are, steatomatous, fibrous, cartilaginous, and calcareous depositions, with a thickened, fragile, and inelastic condition of the arterial coats.

Before describing the depositions, it may be premised that they originate, not in the internal coat, but either in the middle coat, or in the fine cellular tissue interposed between it and the internal coat; that the latter coat can sometimes be peeled off from them in a perfect state, even when they are far advanced; and that the productions themselves are more analogous to those of a fibrous, than of a serous membrane.

The extent, the form, and the thickness of the productions are infinitely various. Sometimes the several species exist separately, but, more commonly, they are found more or less intermingled in the same artery. The most simple morbid alteration is, a loss of elasticity, generally accompanied with increased density and opacity, of the coats of the artery. This state is sufficient of itself to give rise to dilatation, because (as will be more fully explained under the head of dilatation of the aorta) the elasticity and tone of an artery are the powers by which it resists the distending force of the blood.

The next and the most common morbid appearance is that of small, opake, straw-coloured spots, immediately underneath the lining membrane, with slight inequality and corrugation of the membrane around them. At a more advanced period the depositions form considerable, slightly elevated patches, which becoming confluent, sometimes overspread the whole surface. Some of these patches have much the appearance and consistence of bee's-wax, or cheese, though in general their cohesion and flexibility are greater. These are usually denominated steatomatous. Others, presenting nearly the same colour, have a fibrous or ligamentous appearance; while others, again, are more translucent, white, and elastic, like cartilage or fibro-cartilage.

All the depositions described are accompanied with thickening and loss of elasticity of the internal coat, which becomes knotty, wrinkled, and sometimes cracked, scaly, and fimbriated. This state of the internal coat, however, is less marked before earthy depositions have taken place.

These generally commence in the midst of a cartilaginous or fibro-cartilaginous patch, though they are sometimes found in detached scales, and sometimes, in a soft or pasty form, in the midst of cheesy, curdy, or melicerous matter. When they form incrustations, the shape of these is irregularly flattened; the prominences being towards the exterior, rather than the interior of the artery. Their external surface sometimes presents the imprint of the circular fibres of the middle tunic. Their internal surface is sometimes smooth, and evidently covered by the membrane; in other cases it is rough, and the membrane is more or less destroyed. Calcareous depositions are most frequent in the ascending portion and arch of the aorta, but occasionally they pervade the whole of the vessel, and even almost the whole of the arterial system. I saw a case in the Hotel Dieu, in which the great arteries from the heart to the ankle were converted into rigid tubes by ossification, which in parts occupied all the coats and the whole circumference of the vessels. In another case, at St. George's

Hospital, the common iliacs were rigid, and one, which was converted into a bony cylinder, was obliterated by a plug of dense lymph. The arterial system was elsewhere more or less ossified. Both the patients died with gangrenous sores of the legs.

In the arteries at the base of the brain, calcareous and other degenerations are remarkably frequent, and are a principal cause of rupture of the vessels, and apoplectic effusion. It is rare, indeed, to meet with instances of such effusion, exclusive of those from external violence, in which some disease of these arteries may not be detected; and it is remarkable that the disease of the artery is in general connected with hypertrophy of the left ventricle: whence it appears to be a result of over-distention, to which the cerebral arteries are more obnoxious than others, in consequence of their being destitute of a cellular coat, and also of their being ill supported by the pulpy yielding substance of the brain. I have met with several cases of epilepsy which had no other obvious cause than disease of the cerebral arteries. The arteries below the pelvic divarication of the aorta are more frequently ossified than those of the upper extremities and trunk.

Calcareous concretions differ essentially from natural bone. For though some are formed by the secretion of the earthy phosphate in cartilage, even these have not the peculiar fibrous arrangement of bone. But in by far the greater number of cases, the earthy matter is not secreted in any cartilaginous matrix, being simply deposited in the form of an irregular homogeneous crust or crystallization, without any determinate arrangement, and without vitality. The proportion of animal matter in these is very small. Mr. Brande found 100 parts to consist of 65,5 of phosphate of lime and 34,5 of animal matter. In some specimens I have found the quantity of animal matter considerably less.

When ossification is very considerable, it is sometimes attended with induration, inelasticity, and fragility, not only of the internal, but of all the arterial coats; and this state I have seen attended in some cases with thickening, and in others, though less frequently, with attenuation and a horny translucency of the walls of the vessel. The aorta, so affected, generally undergoes dilatation, but very rarely contraction. When the depositions are partial and limited, the internal membrane in the intervals is often perfectly sound. This is especially the case in the ossifications of old people.

It is remarkable that, though morbid depositions are so frequent in the aorta, they are extremely rare in the pulmonary artery. Out of upwards of a thousand cases, in which I have examined this vessel, I have never met with a calcareous deposition in its coats, and only three or four times with cartilaginous and steatomatous disease and dilatation.

Of the causes of morbid depositions in the coats of arteries.—Some authors have considered morbid depositions in the coats of arteries to be, in every case, the various metamorphoses of lymph, effused by inflammation of the acute kind, and of such intensity as always to proclaim itself by obvious symptoms, and require antiphlogistic treatment. Others, again, have supposed that many, if not all, of the depositions in question, take place wholly independent of inflammation of any kind. As principles of treatment of a decided nature have been founded on each of these conflicting doctrines, it is a matter, not of mere speculation, but of practical importance, to examine the subject, and endeavour to ascertain the truth.

Although it would be difficult to deny that acute inflammation may, in some instances, lay the foundation of morbid depositions in the coats of arteries, yet it is certain that they may and do occur independent of it; for they have been found in individuals who had never manifested any symptoms whatever of inflammation, and, even, who had constantly enjoyed the most perfect health.

Analogical evidence derived from other membranes leads to the belief that chronic inflammation is, in most instances at least, the main agent concerned in the production of these depositions. Thus, for example, the dura mater and the pleura are sometimes not only thickened and indurated, but converted into fibrous, cartilaginous, or bony tissue. I have seen the dura mater converted into a calcareous plate nearly as large as the hand, and overspreading one hemisphere of the brain. The preparation was shown to me by my friend Professor Monro, and is in his museum. Mr. Hammick showed me two preparations in his museum of calcareous plates of about two inches in diameter, on the pleura. Changes of this kind are, by general consent, attributed to chronic inflammation; as they are not only found in conjunction with organized adventitious membranes and other anatomical proofs of that form of inflammation, but are often attended with its symptoms. It is to be presumed, therefore, that corresponding changes taking place within an artery are referable to the same cause. That the morbid depositions in the artery should not be exactly identical with those found in other membranes, is to be anticipated on principles of general anatomy; for the effused matter which is the basis of every accidental production, differs in aspect and nature according to the tissue in which it occurs. Thus, as well remarked by Bertin, the cellular tissue and parenchymatous organs secrete pus, properly so called; thus serous membranes secrete a coagulable matter prone to transform itself into cellular or serous layers; thus the periosteum furnishes another matter, which concretes, hardens, and ossifies; thus the arterial tissue, composed essentially of a fibrous membrane, exhales a liquid which hardens, condenses, and becomes converted into catilaginous patches, or calcareous scales.

But admitting the agency of chronic inflammation as a cause of morbid alterations in arteries, there is reason to believe, as before stated, that some of them, particularly the calcareous, may take place independent of it. For they are found in most old people; they sometimes occur in various detached points very remote from each other; they often consist of a simple calcareous scale, or an opake yellow spot, without any morbid state of the surrounding membrane; and such alterations almost always take place without affording the slightest sign, either general or local, of their formation. Now it is scarcely possible to conceive of an inflammation which manifests no symptoms, which is restricted to isolated points often remote from each other, which leaves none of the ordinary vestiges of inflammation in the surrounding parts, and which is the most frequent at that period of life when phlogistic action is the least prevalent. We are brought, then, to inquire what is the cause of morbid depositions when they do not appear to be referable to inflammation.

Here it is necessary to proceed with caution, as the ground is purely speculative. Laennec, indeed, thinks it the most simple and philosophical to acknowledge that we know not the nature of the derangement of the economy which produces an ossification or a cancer, but that very certainly it is not the same as that which produces pus—as inflammation.* If we are not satisfied to remain in this circumspect uncertainty, we can perhaps scarcely venture farther, in the actual state of our knowledge, than to suppose that morbid productions are sometimes results of a depraved action of the vessels not identical with, or not amounting to inflammation—a doctrine, indeed, which rests on the basis of sound observation, and which has been extensively received since the accurate researches of the present century have bred a "philosophic doubt" on the tenet of the ancients, that all accidental productions are the effects of inflammation. Admitting a depraved action of the vessels, it is rational to suppose that, like inflammation, it would derive its particular character from the tissue which it affects: hence, that the fibrous and fibro-serous tissues of the arteries might degenerate into cartilage, bone, &c.—the changes to which those tissues are prone under the influence of inflammation.

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* De l'Auscult. tom. ii. p. 684.

But what is it that calls this depraved action into activity? It appears to me that over-distention of the arteries by the force of the circulation is what, principally at least, produces the effect. To this opinion I am led by the following consider ations: that arterial ossifications are the most common when the left ventricle is hypertrophous: that (according to an observation of Boerhaave related by Morgagni) they are found in stags long and often exercised in running, and not in those which lead a tranquil life in the parks of the great: that diseases of arteries and aneurism are more common, in the proportion of at least seven or eight to one, in men than in women, the life of the former being much more laborious, and the circulation more liable to excitement from potation of vinous or spirituous liquors, &c. that ossifications, &c. occur in those arteries more especially, which are most exposed to over-distention; namely, the arch of the aorta, which immediately sustains the whole brunt of the left ventricular contraction, and the arteries of the brain, which, not having the support of a cellular sheath and being bedded in a soft pulpy substance, are weaker than any others: that they are more especially incident to the aged, in whom the arterial and all other tissues sustain a diminution of elasticity and cohesiveness in consequence of the diminished vascularity which characterises old age. Perhaps the same reason, *viz.* over-distention, may be assigned for the remarkable frequency of the arterial depositions in those who have suffered much from syphilis or mercury; for as these maladies induce a cachectic state which lessens the elasticity of all the tissues, the arterial tissue would, under these circumstances, suffer proportionably more from the distending pressure of the circulation. To the above catalogue we may perhaps add gout; (an affection which is remarkably often attended with ossifications and their frequent concomitant, angina pectoris;) for the gouty habit is in general accompanied with a morbid degree of plethora, and consequently with over tension of the arterial system.

It may be remarked, finally, that even they who attribute all the morbid alterations in arteries to inflammation, admit the necessity of *specific* inflammations to account for those alterations which are not normal products of ordinary inflammation. Thus Kreysig thinks that calcareous incrustations are only produced by gouty inflammation; and Bouillaud, who employs the word inflammation in perhaps too extended a sense, believes that there must be a *peculiar predisposition* for each effect of the one same cause,—that every variety of deposition must be the result of a different specific inflammatory action.

SECTION III.

SYMPTOMS, DIAGNOSIS, PROGNOSIS AND TREATMENT OF ARTERITIS.

Diagnosis difficult, 168. *Symptoms of* Acute *Arteritis enumerated*, 169. *Aortic and arterial throbbing*, 169. *Effects of Inflammation of the Internal Membrane of the Heart on its Action*, 170. *Causes protracted Convalescence in Fever*, 170. *Fallacies to which the above Symptoms are liable*, 171. 1*st. Those from inflammatory Complications*, 171. 2*d. Those from Complications not inflammatory, but producing arterial throbbing*, 171. *viz.* A. *Disease or Dilatation of the Aorta—Mode in which it occasions throbbing explained*, 171. B. *Nervous irritability*, 172. C. *Reaction from loss of Blood*, 172. D. *Adhesion of the Pericardium*, 173. E. *Tumors, Serous Effusions, &c.* 173. *Symptoms of* Chronic *Arteritis*, 174. Prognosis, 174. Treatment, 175.

Symptoms and diagnosis of acute arteritis.—The diagnosis of arteritis is one of the most difficult in the range of medicine; as the malady presents no signs that are peculiar to itself, and that distinguish it from other diseases.

I shall first describe the signs by which some authors have imagined that they could detect acute inflammation in the aorta, and then subjoin the sources of fallacy to which, according to my experience, those signs are liable. The practitioner, being thus warned against the various causes of deception, will be better able to give their full value to the signs themselves; and he may in this way, I think, make out the diagnosis with some success.

The principal sign of acute inflammation in the aorta, according to authors, is, a pulsation of the aorta much more violent than in the healthy state. Pain and a sensation of heat in the region of the aorta, with anxiety and faintishness, are symptoms of a less constant nature, but which, nevertheless, it is important to mark. To these are to be added acceleration of pulse, heat and dryness of the skin, thirst, anorexia, and all the other concomitants of phlogistic action.

The position of the thoracic aorta is such that the strength of its pulse can only be explored in the hollow at the summit of the sternum and above the clavicles: the beating of the ventral aorta may be more easily examined; namely, by applying the hand, or, what is better, the stethoscope, on the abdomen.

Violent pulsation of the aorta from inflammation is often accompanied with a similar pulsation in any or all of the great arteries, the reason of which is, that the inflammation not unfrequently extends to other arteries, and even to the whole of the arterial system.

With respect to inflammation of the internal membrane of the heart, it is excessively rare in an isolated form, being almost invariably attended either with carditis or pericarditis. Analogy indicates that it ought to be characterized by augmentation of the force and frequency of the action of the heart and arteries, and leads us to expect that when the inflammation reaches and disorganizes the muscular substance, it would render it incapable of contracting with its natural strength and regularity. Observation confirms what analogy points out. In fevers properly so called, which constantly appear to be accompanied with an irritation of the heart, the frequency and the force of the pulse are the two principal phenomena that strike our senses; but should the fever assume a typhoid type, and give rise to an irritation of the heart, so profound that the muscular substance itself is attacked, the pulse loses its force and regularity while it augments in frequency, and an unexpected death often terminates this always formidable complication. Should the patient recover, the slowness of his convalescence and the frequency of pulse which characterizes it, are probably dependent either on an irritable state of the internal membrane left by the inflammation, or on softening of the muscular substance,—a subject to which I shall revert when treating of softening.

Such are the symptoms of acute arteritis assigned by authors. The fallacies to which they are liable are so numerous, that many able practitioners formally avow their inability to make the diagnosis. The fallacies arise, 1st. from the inflammatory complications with which arteritis—and more particularly aortitis, is accompanied: 2d. from affections which, though unattended with arteritis, occasion arterial pulsation.

1. *The inflammatory complications of aortitis.*—Acute aortitis is very rarely known to exist (so far as we can judge from its anatomical vestiges—the only positive criteria) independent either of continued fever, or of inflammation of some of the principal viscera, particularly the lungs, pleura, and pericardium. In these complicated cases the symptoms of aortitis are lost in those of the concomitant malady, which is of itself capable of producing, to a greater or less extent, the same violent arterial pulsation, the same anxiety and faintishness as are considered characteristic of aortitis.

2. *Affections which, though unattended with arteritis, occasion arterial pulsation.*

A. When the interior of the ascending portion, or arch of the aorta, is ossified or cartilaginous, there is generally a preternatural pulsation above the clavicles, and this always takes place when the artery is dilated. For, as the blood permeates the diseased or dilated portions with greater difficulty than others in consequence of its being reverberated in counter-currents and eddies from prominencies in the vessel, those portions sustain, at each contraction of the heart, an increase of the expanding force of the blood, the lateral pressure of which is always augmented in proportion as the direct current is impeded. The mode of discriminating pulsation proceeding from disease and dilatation of the aorta, will be fully explained in the article on dilatation of the aorta.

B. Persons of an irritable temperament, especially delicate, hysterical females and hypochondriacal males, are subject to morbid arterial pulsation, sometimes confined to the aorta alone, and sometimes universal throughout the system. When it is seated in the descending aorta, the patient complains of " a fluttering," in the epigastrium.*

C. Morbid arterial pulsation is a phenomenon of reaction after loss of blood—a fact to which the attention of the profession has been drawn with great ability by Dr. Marshall Hall, and which I have verified, not only by observations on the human subject, but by extensive experiments on animals, performed in concert with that gentleman. The pulsation not unfrequently supervenes, and subsists for a considerable time, after the free depletions necessary for the cure of acute inflammatory affections, as pleuritis, pericarditis, peripneumony, phrenitis, peritonitis, &c. In these cases it would be a dangerous error to suppose that the pulsation depended upon aortitis.

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* See Nervous Pulsation of the Aorta; also the Explanation of it, p. 78.

D. I have found arterial pulsations to accompany adhesion of the pericardium, especially for the first year or so after the attack of pericarditis which occasioned it. The phenomenon is attributable to the jerking and, as it were, spasmodic manner in which the heart contracts under such circumstances. This subject will be noticed in the article on pericarditis with adhesion of the pericardium. It may be remarked that there was adhesion of the pericardium in two of the three cases from which more especially Bertin and Bouillaud derive their opinion that aortic pulsation is always a sign of aortitis.*

E. Tumours, serous effusions, &c. in contact with the aorta, give it the character of preternatural pulsation, as they transmit its impulse more strongly than the parts which properly environ it. The mode of distinguishing these pulsations is treated of under the diagnosis of aneurisms of the aorta.

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* Bertin Obs. xxvii, xxviii, and xxix.

From this long catalogue of fallacies it will be apparent that the diagnosis of arteritis is beset with much difficulty. Still, it is not wholly hopeless; for, by a negative process—by ascertaining that the pulsation is not attributable to any of the causes mentioned, I believe it is possible to form an opinion which will not be very wide of the truth.

Of *chronic* arteritis it is sufficient to say that, though it in all probability deteriorates the general health, it presents no distinctly appreciable signs but those of the structural alterations—the depositions, dilatations and valvular obstructions—to which it gives rise. These signs are treated of under the heads of aneurism of the aorta and valvular disease.

Prognosis.—Arteritis, unless very extensive, is not a dangerous disease by its immediate effects; but it may lay the foundation of disease of the arterial coats and of the valves of the heart, which may ultimately prove formidable by inducing aneurism of the vessels, and disease of the heart. When arteritis is complicated with fevers, or visceral inflammations, the prognosis must be founded on the symptoms of the latter maladies.

Treatment of arteritis and inflammation of the internal membrane of the heart.—Arteritis is to be treated on the same general principles as any other inflammation; namely, by bleeding, purgatives, diaphoretics, low diet, cooling diluent drinks and perfect tranquillity. The latter is absolutely necessary, as any efforts which accelerate the circulation, increase the tension, and therefore the irritation, of the arterial system. On the same principle remedies which calm the circulation, as digitalis, tartrate of antimony, superacetate of lead, hydrocyanic acid, are useful auxiliaries when discreetly employed; but they should not supersede the more important remedies above mentioned, nor should they be given at a late period of the disease, when the symptoms, which of themselves become complicated, should be kept as unembarrassed and clear as possible. When mercury is not contra-indicated by a strumous or shattered constitution, its exhibition to the extent of slight but prompt salivation, after the first violence of inflammatory action has been repressed, is attended with excellent effects. (See treatment of pericarditis.)

When arteritis is complicated with visceral inflammation or fever, the treatment must be directed to the primary malady.

PART III.

ORGANIC AFFECTIONS OF THE HEART AND GREAT VESSELS.

THIS part will comprise, first, the diseases of the muscular substance, and, secondly, those of the internal membrane and valves, and of the aorta. The organic diseases of the external membrane or pericardium are comprised in Part II. for reasons there assigned. (See Adhesion of the Pericardium.)

CHAPTER I.

HYPERTROPHY OF THE HEART.

SECTION I.

ANATOMICAL CHARACTERS WITH CLASSIFICATION AND NOMENCLATURE OF HYPERTROPHY.

Definition, 177. *Imperfections of the old Classification and Nomenclature*, 177. *New Classification*, 178; *and Nomenclature*, 179. *Natural Dimensions and Proportions of the Heart*, 180. *Anatomical Characters of Hypertro-*

phy, 180. *Muscular Substance reddened and indurated, Hypertrophy with Dilitation—Universal*, 182. Of left Ventricle, 183. *Degree of Thickening and Situation of the greatest*, 183. *Hypertrophy with Contraction*, 184. *Hypertrophy of Columnæ Carneæ*, 184. *Of the Septum*, 184; Of right Ventricle, 184. *The Hypertrophy is principally in the Columnæ Carneæ—Curious Effect sometimes produced*, 184. *Degree of Thickening*, 185. *Hypertrophy sometimes partial in a Cavity, and even* Conjoined *with Attenuation*, 185. Of the Auricles, 186.

HYPERTROPHY is an augmentation of the muscular substance of the heart, resulting from increased nutrition.

As late as the year 1811, this affection was very imperfectly understood. No other form of it had been recognised, than that which was denominated by Corvisart *Active Aneurism*, (the *hypertrophy with dilatation* of Laennec,) a combination of two distinct affections which may exist independently of each other. Morgagni,* Corvisart,† and Burserius,‡ indeed, had each seen and described hypertrophy *without* dilatation; but it had not particularly arrested their attention, nor led to any inferences. It was reserved for M. Bertin in 1811 to throw new light on this subject. In three memoirs presented to the Académie Royale des Sciences he proved, that hypertrophy might exist, not only with dilatation, but also without it; that is, with a natural, and even with a diminished size of the cavity. Since that epoch, the concurrent observations of other pathologists, both abroad and in this country have confirmed the accuracy of his observations, and led to the substitution of a new and more definite classification and nomenclature, in place of the inaccurate distinctions into *Active* and *Passive Aneurism* introduced by Corvisart.

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* Epist. xvii. art. 21.

† Edit. 3. p. 335.

‡ Instit. Med.

Hypertrophy presents the following varieties.

1. *Simple hypertrophy,* in which the walls are thickened, the cavity retaining its natural dimensions.

2. *Hypertrophy with dilatation.* This, the *excentric* or *aneurismal hypertrophy* of Bertin, presents two varieties: viz.

A.—With the walls thickened and the cavity dilated.

B.—With the walls of natural thickness and the cavity dilated: i. e. *hypertrophy by increased extent of the walls.*

3. *Hypertrophy with contraction.* In this the *concentric hypertrophy* of Bertin, the walls are thickened and the cavity is diminished.

This classification is no less convenient than conformable to nature. The form B. of the second variety was not known to Laennec, though it was to Bertin. That it *really* consists of an augmentation of muscular substance, and therefore constitutes hypertrophy, is too manifest to require comment; but a further proof than mere structure, is, that it sometimes produces the symptoms of hypertrophy, —a fact which the writer ascertained and made known several years ago before he had any knowledge that M. Bertin had done the same.*

The terms "*excentric* or *aneurismal*," and "*concentric*" are not so simple and expressive as *hypertrophy with dilatation* introduced by Laennec, and its natural converse *hypertrophy with contraction*. There is a further objection to the nomenclature of Bertin. His first variety of Dilatation, identical in its nature with his second variety of hypertrophy, is designated by a totally different name, viz, *active aneurism;* (Bertin p. 376) which could scarcely fail to lead the inexperienced student into the erroneous idea, that there was a difference in the nature of the two affections. Now the only difference consists in degree—in a predominance of the one state over the other. The terms, therefore, should be such as distinctly to imply identity in nature, and difference in degree only; and this is done in the simplest manner by giving precedence to the word hypertrophy, or dilatation, according as the one affection or the other predominates. Thus *hypertrophy with dila-*

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* Vid. an Essay by the writer in 1824, read to the Royal Med. Soc. Ed.

tation denotes a predominance of hypertrophy, while the converse *dilatation with thickening* (vid. dilatation) denotes a predominance of dilatation. *Hypertrophy by increased extent* (without altered thickness) *of the walls*,—the form B. of the second variety, is thus designated when it is accompanied with the symptoms of hypertrophy; but it is called *simple dilatation* when the symptoms are those of dilatation.

I have thought it necessary to speak thus particularly on the subject of nomenclature, as, up to the present moment, it has created much confusion, and must continue to do so until the terms *active* and *passive aneurism* are forgotten.

Anatomical characters of hypertrophy.—Before describing the anatomical characters of hypertrophy of the heart it is necessary to give the reader an idea of the natural dimensions of this organ. Unfortunately, it is impossible to determine these positively; for, as they vary according to age, sex, and other circumstances, there is no immutable standard of comparison which might serve as a criterion. It is only by the eye, therefore, (and an experienced eye is necessary for the purpose) that it can be determined whether the proportion of the heart to the system, and of its several parts to each other are natural. The proportions assigned by Laennec, approach perhaps as near the truth as it is possible to arrive—they are as follows. " The heart, comprising the auricles, ought to have a size equal to, a little less, or a very little larger than, the fist of the subject. The walls of the left ventricle ought to have a thickness a little more than double that of the walls of the right: they ought not to collapse when an incision is made into the cavity. The right ventricle, a little larger than the left, and having larger columnæ carneæ notwithstanding the inferior thickness of its walls, ought to collapse after an incision has been made into it. Reason indicates and observation proves, that, in a sound and well built subject, the four cavities of the heart are, within very little, equal to each other. But as the walls of the auricles are very thin, and those of the ventricles have much thickness, it results that the auricles form scarcely a third of the total volume of the organ, or the half of that of the ventricles." In the fœtus and very young children, the thickness of the left ventricle does not exceed that of the right to the extent described.

The right cavities are rather larger than the left, and this is not owing to sanguineous distention attendant on dissolution: for the disparity is found, though in a less degree, in animals destroyed by hæmorrhage.

The muscular substance in hypertrophy is usually firmer and redder than natural. These characters, however, are not essential to the disease; and, when they exist in a great degree, they constitute

Induration, a distinct affection, dependent, not on increased, but rather on *altered* nutrition of the part.

Hypertrophy may be confined to a single cavity, or it may affect several, and even the whole simultaneously. Sometimes one cavity is thickened, whilst another is attenuated. The full consideration of this subject comes under the head of exciting causes; as it is principally by these, that the nature and extent of the affection is determined. It may here suffice to remark, generally, that the ventricles are more obnoxious to the disease than the auricles, because they are exposed to a greater variety of exciting causes, and because the auricles are remarkably protected by the auriculo-ventricular valves.

When all the cavities are hypertrophous and at the same time dilated, the heart attains a volume, two, three, and occasionally even four times greater than natural, its form, instead of being oblong, is spherical, its apex is scarcely distinguishable, and, as the diaphragm does not retire sufficiently to yield space downwards for the enlarged organ, it assumes an unnaturally horizontal position, encroaching so far upon the left cavity of the chest, as sometimes to force the lung upwards as high as the level of the fourth rib, or even higher. I lately examined a subject in which it had been forced much higher. When great enlargement is accompanied by adhesion of the pericardium, the organ is secured by the attachments of the membrane, in a higher situation than its gravity would otherwise dispose it to assume; and being thus impacted between the spine and the anterior parietes of the chest, it is apt to occasion a preternatural prominence of the præcordial region. I am not aware that this remark has been made by any other writer, but I have seen the phenomenon in so many instances that I am disposed to assume it as a general fact (see adhesion of the pericardium).

The left ventricle, being more prone to thickening, and not less to dilatation than the right, sometimes attains a volume seldom or never acquired by the right; and, when its enlargement is enormous, it occupies not only the left præcordial region, but extends far under the sternum, where its impulse and sound may be mistaken for those of the right ventricle.* (Case of Lambert).

The walls of the left ventricle, the natural thickness of which averages about half an inch in the adult, may be increased to the extent of one, one and a half, or, according to some, of two inches. The cases are rare in which it exceeds an inch and a quarter. The situation of the greatest thickening is usually a little above the middle of the ventricle, where the columnæ carneæ take their origin.

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* Laennec, tom. ii. p. 507.

Thence, the thickness decreases rather suddenly towards the aortic orifice, and gradually towards the apex, where it is reduced to less than half. When hypertrophy maintains these proportions in the different parts of the ventricle, the state is only an exaggeration of the natural form. The case is different when the hypertrophy takes place inwards and diminishes the cavity; for then the whole ventricle is nearly equally thickened and its form is unusually globular.

The columnæ carneæ generally participate in hypertrophy, but sometimes, when there is much dilatation also, they appear to be stretched, flattened and attenuated. The inter-ventricular septum, though belonging almost entirely to the left ventricle, is commonly less thickened than the external walls. When the left ventricle is greatly enlarged, the right, if unchanged, is applied in a flattened form, to its superior and lateral part, and by contrast looks singularly small. But if, as generally happens, the right is elongated, it is, as it were, folded around the left.

When the *right ventricle* alone is hypertrophous it may descend lower than the left and constitute the apex of the heart. Its columnæ carneæ, naturally more numerous and complicated than those of the left, are more susceptible of thickening than the walls themselves of the cavity. Hence, the increased size of the columnæ is commonly the first object that arrests the attention, and to them alone is the hypertrophy in many instances confined. They are sometimes so curiously interlaced and attached, as to traverse the ventricle in every direction, subdivide it into various compartments, and in some cases, almost totally to fill up its cavity. (As in case 89 by Bertin, and that of Collins.) These changes never take place to the same extent in the left ventricle. The total thickness of the walls of the right ventricle, naturally averaging three lines, rarely exceeds four or five; yet it has been known to attain from eleven to sixteen, as appears from the 88th case of Bertin, and one, by Soins, in the *Archives de Médiçine.* In a girl of nine years old (see case of Collins—Cyanosis) I have met with it measuring six or seven lines; which is equal in proportion to nearly double that extent in the adult. Hypertrophy without dilatation is much more rare in the right than in the left ventricle. The greatest thickening of the right ventricle is near its base: lower down, though the columnæ carneæ be enlarged, their interstices are usually thin, and not unfrequently translucent.

Hypertrophy may not only be confined to a single ventricle, whether it be the right or the left, but it may be confined to particular parts only, as the base, the septum, the apex, the columnæ carneæ, or the external walls; the remainder of the cavity being either natural, or attenuated. Again, a thickened ventricle may be contracted in one part, while it is dilated in another. In examining in the dead subject mixed cases of these descriptions, it is necessary to counterpoise the opposite conditions, to balance the hypertrophy against the extenuation, and the dilatation against the contraction, in order to determine which is the predominant affection.

The hypertrophy of the auricles is almost invariably of the second species or that with dilatation. Laennec even states that he has never met with any other.* The *simple*, and the *contracted* forms, however, are not without example. The thickening is diffused in a very uniform manner throughout the cavities, the musculi pectinali being the only parts in which it is more considerable than elsewhere; and, as they are larger and more numerous in the right, than in the left auricle, it is in the former that hypertrophy proceeds to the greatest extent. It occasionally renders the auricle nearly as thick as the right ventricle. This I have never known to take place in the left auricle. Sometimes the musculi pectinali are the only parts in which hypertrophy shows itself. The thickening of the auricular walls seldom exceeds double the natural state, and, being even then inconsiderable, it may easily be overlooked by an inexperienced eye. When it amounts to a quarter of an inch, which is rarely the case, it is very perceptible.

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* Laennec de l'Auscult. tom. ii. p. 524.

SECTION II.

MODE OF FORMATION WITH THE PREDISPOSING AND EXCITING CAUSES OF HYPERTROPHY.

Hyper-nutrition how occasioned in Muscles in general, 187. *How in the Heart,* 188. *Why the stronger Cavities are more predisposed to Dilatation than the weaker,* 189. *Dilatation may take place simultaneously with Hypertrophy and vice versâ,* 190. *Causes of Hypertrophy in the right Ventricle,* 190. *Objection to M. Bertin's Opinion that the Presence of Arterial Blood is the Cause of Hypertrophy,* 191. Exciting Causes *of Hypertrophy. 1st. Nervous,* 192. *2d. Physical,* 193.—*Those which accelerate the Circulation,* 193.—*Those which retard it,* 193. *Young Plethoric Persons most susceptible of their influence,* 194.

Mode of formation and predisposing causes of hypertrophy.—Hypertrophy takes place in the heart by the same process as in any other muscle. Increased action causes an augmented afflux of blood and there results a corresponding increase of nutrition. Diminished action, on the contrary, has the reverse effect. Thus, the arms of the smith and the legs of the dancer, are unusually robust; while limbs paralysed or not exercised, are pale and emaciated. If, however, the circulation can be reinvigorated in the palsied part, nutrition is increased. An individual within my knowledge, whose arm had, in consequence of an attack of heuriplegia, been for twenty years emaciated, contracted, without radial pulse, and immoveably fixed to the side, submitted the limb to the process of vigorous shampooing. In a few months, the pulse returned, the emaciation sensibly diminished, and the motive power was so far restored that the individual could raise the hand above the head.

In the same way, when, from mechanical obstruction or any other cause, blood is inordinately accumulated in the heart, the organ is provoked to extraordinary efforts; it struggles against the obstacle; it frets and labours to overcome it; the coronary arteries are excited to increased activity; augmented nutrition ensues; the parietes are thickened, the muscular power is increased; the effects, superadded to the cause, induce a still greater violence of action; and, thus, the disease is not only established, but has a constant tendency to increase.

The left ventricle is much more prone to hypertrophy than the right; and the right, again, than the auricles.

This admits of explanation on very simple principles. It is found that hollow muscles resist over-distention by their contents with a force exactly proportionate to their strength. Now, as the act of resistance, by stimulating the arteries to increased action, is the cause of increased nutrition, it follows that stronger muscles must be the more susceptible of hypertrophy. Accordingly, on referring to the heart, we find that the relative structure of its several compartments is such as to predispose the organ to those changes which it actually undergoes from over-distention.

The left ventricle, for example, being charged with the immense burden of the greater circulation, is proportionably substantial and robust; the right, having the comparatively light task of propelling the blood through the minor or pulmonary system, is little more than one third as thick and powerful as the left: the auricles, again, having a still less laborious function to perform, have a still more limited muscular provision.

Hence, it is easily understood how a distending force sufficient to overcome the contractile and elastic power of the right ventricle, might merely operate as a stimulus to the superior muscularity of the left. While the former, therefore, incapable of reacting on its contents, would dilate; the latter, excited to extraordinary efforts, would become hypertrophous.

It is not, however, to be supposed, that while the left ventricle is becoming hypertrophous, it may not, at the same time, undergo dilatation: nor, on the other hand, that the right ventricle, while yielding to dilatation, may not become hypertrophous; for observation teaches us, that the combination of hypertrophy with dilatation, either in the left ventricle alone, or in the two conjointly, is the most ordinary form of organic disease of the heart.

For an explanation of the cause why dilatation accompanies hypertrophy, the reader may refer to the chapter on dilatation. Why hypertrophy sometimes accompanies dilatation of the right ventricle, may be here explained, and it admits of an explanation in one or other of two ways. 1st. It has been remarked by Laennec,* that a large proportion of mankind are born with ill proportioned hearts, the parietes being a little too thin, or a little too thick on one or both sides. Now when this unnatural thickness exists in the right ventricle, it is clear from what has been said above, that it must impart to that ventricle an increased disposition to hypertrophy. This explanation, however, is not very satisfactory, as the existence of the malformation described by Laennec cannot be positively proved: yet, as all the other organs and parts of the body are liable to defects of natural conformation, it is consistent with analogy to suppose that the heart may be liable to the same.

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* Traité de l'Auscult. tom. ii. p. 496.

2dly. As augmented nutrition is excited in the left ventricle by stimulating it in proportion to its power, so a stimulus bearing the same proportion to the power of the right ventricle, must have the same effect on it also. Accordingly, in the majority of cases of hypertrophy of the right ventricle, an obstacle is found to exist of such a nature as the one described. The obstacles which I have most frequently found to produce the effect, are, contraction of the mitral valve operating in a retrograde direction through the lungs, and that of the semi-lunar valves of the pulmonary artery. These affections being usually slight at their commencement and slow in their progress, oppose an obstacle to the circulation not only moderate in degree, but constant in its operation,—the two circumstances best calculated to induce hypertrophy of the right ventricle.

M. Bertin conceives that the greater tendency of the left ventricle than of the right to hypertrophy, depends upon the more stimulant quality of the arterial blood circulating through the former. This opinion he founds on the circumstance that hypertrophy of the right ventricle in most cases accompanies patescence of the foramen ovale, which lesion he thinks causes an influx of arterial blood into the right ventricle. But, admitting that arterial blood in the right ventricle does occasion hypertrophy, it does not follow that it should have the same effect on the left; for, of the former ventricle it is a morbid stimulus, but of the latter it is the natural one. Accordingly, direct proof is to be found in the auricles that arterial blood is not the cause of hypertrophy; for the left auricle, which on M. Bertin's principle ought to be more subject to hypertrophy than the right, is less so. It will be shown, moreover, in the chapter on malformations of the heart, that, in the cases on which M. Bertin founds his opinion, the blood does not enter the right ventricle.

Exciting causes of hypertrophy.—According to the foregoing opinions on the mode of formation of hypertrophy, it will be apparent that every circumstance capable of increasing the action of the heart for a sufficient length of time, may be a cause of hypertrophy. These circumstances may be either, first, of a nervous, or second, of a mechanical nature.

The former class comprises all moral affections and all derangements of the nervous function that excite long continued palpitation. To these I would add protracted rheumatic fevers; for I have known these give rise to hypertrophy though there was apparently no inflammation of the heart or its membranes.

The latter class embraces all physical causes which can either accelerate, or obstruct the circulation, and thus occasion a preternatural pressure of the blood upon the heart.

The physical causes which accelerate the circulation, are, violent and protracted corporeal efforts of every description. In growing youths, excessive rowing is one of the most efficient. I have met with several instances in which it has produced the effect.

The physical causes which obstruct the circulation are very numerous. They comprise smallness of the aorta, whether congenital or acquired; dilatation of the aorta; inequalities of its internal surface; all diseases of the valves of the heart which either contract their apertures or impede their movements; adhesion of the pericardium; all affections of the chest that obstruct the circulation through the lungs; as peripneumony, acute or chronic; empyema; hydrothorax; chronic catarrh; emphysema; phthisis;* narrowness of the chest, either congenital, or occasioned by curvature* of the spine &c.; encroachment of the diaphragm on the cavity of the chest from the pressure of tight stays, of the gavid uterus, of abdominal dropsy, aneurism, &c.

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* I have not found that phthisis is so decided a cause of disease of the heart as we should be led to suppose from the extreme pulmonary obstruction to which it sometimes gives rise. The reason of this appears to me to be, that, in the early

In reference both to the nervous, and the physical causes of palpitation, it may be said that young persons of a plethoric habit and sanguine temperament are the most susceptible of their influence. Hence it is that very stout and high coloured females, from the age of seventeen to twenty-five are peculiarly subject to hypertrophy. I have noticed this fact in reference more especially to servants coming from the country.

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stages, when the disorganization is not extensive, the circulation is little embarrassed; and in the advanced stages, the mass of circulating fluids is so much diminished in consequence of deficient nutrition and augmented cutaneous transpiration that the heart sustains little additional burden from the obstruction in the lungs. In most cases, however, the right ventricle is found somewhat dilated,—a remark which has been made more especially by M. Louis and Dr. Williams.

[*] The majority of hump-backed persons are ultimately attacked by disease of the heart.

SECTION III.

ORDER OF SUCCESSION IN WHICH THE SEVERAL COMPARTMENTS OF THE HEART ARE RENDERED HYPERTROPHOUS BY AN OBSTACLE BEFORE THEM IN THE COURSE OF THE CIRCULATION.

Why the left Ventricle suffers first and most from an Obstacle in the Aorta or Arterial System, 196. *Why the left Auricle is seldom affected,* 196. *The right Auricle and Ventricle suffer in the same way from an Obstruction of the Lungs,* 196. *Which Cavities suffer when the Obstruction extends to all,* 196. *Contraction of the mitral Orifice produces Emaciation of the left Ventricle, Hypertrophy of the left Auricle, and Retrograde Obstruction,* 197. *This reaching the right Ventricle causes its Hypertrophy,* 197. *These Dispositions of Parts the most frequent Cause of Pulmonary Apoplexy—Why,* 197. *Permanent Patescence of the Mitral Orifice causes Hypertrophy of the left Auricle and retrograde Obstruction,* 198. *When the Obstacle is primitively in the Lungs, the right Ventricle suffers first—then the Auricle,* 198. *An Impediment to the Return of the Venous Blood into the Heart causes universal Venous Retardation, finally propagated through the Capillaries to the Arterial System and ultimately in a Circle to the Heart,* 198. *Thus Hypertrophy of a Cavity may be occasioned by an Obstruction behind it,* 199. *Whether Hypertrophy, Dilatation, or both be produced by an Obstacle, depends on the Relation between the Force of the Obstacle and the Power of Resistance,* 199. *Permanent Congestion causes Dilatation —mere Resistance, Hypertrophy,* 199. *List of the Varieties of Hypertrophy in the Order of their Frequency,* 200.

As an obstacle to the circulation operates on the heart in a retrograde direction, the cavity situated immediately behind it is the first to suffer from its influence. Accordingly all the impediments seated in the aorta, its mouth, or the arterial system, act primarily on the left ventricle, which being likewise exposed to the heaviest burden when the circulation is accelerated, has to conflict against a greater variety of exciting causes of hypertrophy, than any other cavity of the heart. On this account, therefore, as well as from the thickness of its parietes, it is subject to hypertrophy in a greater degree than any other.

So long as the left ventricle is capable of propelling its contents, the corresponding auricle, being protected by its valve, remains secure. Hence, in a large majority of cases, the auricle is perfectly exempt from disease, while the ventricle is even enormously thickened and dilated. But when the distending pressure of the blood preponderates over the power of the ventricle, its contents, from not being duly expelled, constitute an obstacle to the transmission of the auricular blood. Hence the auricle becomes over distended, and the obstruction may be propagated backwards through the lungs to the right side of the heart, and there occasion the same series of phenomena.

When the obstruction thus becomes universal, as is frequently the case, it may either happen that all the cavities are thickened, or those only which, from their conformation, have the greatest predisposition to it.

When the mitral orifice is contracted, especially if the aperture be very small, the left ventricle, being insufficiently supplied with blood, is not stimulated to its ordinary contractile action, and consequently becomes emaciated and occasionally flaccid or softened. Meanwhile, the left auricle, having to struggle against the contracted valve in front, and also to sustain the distending pressure of the blood flowing in from the lungs, invariably becomes thickened and dilated. The engorgement, extending backwards through the lungs to the right ventricle, occasions its hypertrophy and dilatation; under which circumstances, namely, hypertrophy of the right ventricle and contraction of the mitral valve, the lungs suffer in a pre-eminent degree: for, being exposed to the augmented impulsive power of the right ventricle behind, and incapable of unloading themselves on account of the straitened orifice in front, their delicate and ill-supported vessels are strained beyond the power of resistance. If, therefore, they cannot disgorge themselves sufficiently by a copious secretion of watery mucus, they effuse blood by transudation into the air-vesicles and tubes, and form the disease denominated *pulmonary apoplexy*. I have found this affection to occur more frequently under the circumstances described, than under any other.

When the mitral orifice is permanently patescent, so that, at each ventricular contraction, blood regurgitates into the auricle, this cavity suffers in a remarkable degree: for it is not only gorged with the blood which it cannot transmit, but, in addition, sustains the pressure of the ventricular contraction. Permanent patescence of the mitral orifice, therefore, constitutes an obstruction on the left side of the heart, and the effect of this, as of contraction of the orifice, may be propagated backwards to the right side.

When the impediment to the circulation is primitively seated in the lungs, the right ventricle, situated immediately behind them, is the first to experience its influence; and when the cavity is so far overpowered by the distending pressure of the blood as to be incapable of adequately expelling its contents, the obstruction extends to the auricle, —the process being exactly the same as that which I have already described (p. 196) in reference to the left ventricle and auricle.

Obstruction in the right auricle, whether from this or any other cause, presents an obstacle to the return of the venous blood and therefore ultimately causes retardation throughout the whole venous system. Nor is this all; for the retardation is propagated through the capillaries to the arterial system and thus at length returns in a circle to the heart. In this way is explained what at first sight appears an anomaly: namely, that the left cavities are sometimes rendered hypertrophous by an obstruction situated behind them in the course of the circulation, as, for instance, when the left ventricle is rendered hypertrophous by a contraction of the mitral orifice.

The reader must here be again reminded that the exciting causes of hypertrophy are equally those of dilatation; and that, supposing no unknown agencies to interfere, as may sometimes possibly happen, it depends on the proportion which the cause bears to the reacting energy of the cavity exposed to its influence, whether that cavity become affected with hypertrophy, with dilatation, or with a combination of the two.

It may be said, generally, that when congestion is *constant* in a cavity, dilatation is more commonly the result; and that when there is only resistance to the expulsion of the blood, without constant engorgement of the cavity, it is more common for hypertrophy to be produced. Contraction, for instance, of the aortic orifice causes hypertrophy of the left ventricle in a greater degree than dilatation; whereas, patescence of that orifice, attended with regurgitation and constant engorgement of the cavity, causes dilatation in a greater degree than hypertrophy.

Hypertrophy with contraction most commonly proceeds from straitening of an orifice. Thus the greatest hypertrophy with contraction of the right ventricle upon record was accompanied with straitening of the pulmonary orifice to two lines and a half in diameter. Case 87 by M. Bertin. I have met with a very similar case, and several connected with malformation of the heart are on record.

It may be useful to subjoin a list of the various forms and combinations of hypertrophy and dilatation, and to shew the comparative frequency of their occurrence. On the latter point I shall offer the results of my own observation, and I believe that they correspond very closely with those of others.

The diseases are of more frequent occurrence in proportion as they are higher in the following scale.

1. Hypertrophy with dilatation of the left ventricle and a less degree of the same in the right.

2. Hypertrophy with dilatation of one ventricle, especially the left, with simple dilatation of the other.

3. Simple dilatation of both ventricles.

4. Simple hypertrophy of the left and hypertrophy with dilatation of the right.

5. Dilatation with attenuation of the left.

6. Hypertrophy with contraction of the left.

7. Hypertrophy with contraction of the right.

Of the Auricles.

1. Distention, particularly of the right, from congestion during the period of dissolution.

2. Dilatation with hypertrophy.

3. Simple hypertrophy.

4. Hypertrophy with contraction.

SECTION IV.

PATHOLOGICAL EFFECTS OF HYPERTROPHY AND MODE OF THEIR PRODUCTION.

Views of Laennec and his Predecessors imperfect, 202. *Those of Bertin better*, 203;—*but partly inaccurate*, 204. *How Œdema of the Lungs, Dyspnœa and Retardation of the Venous Circulation are produced by Hypertrophy*, 205. *Why Dropsy is produced less readily by Hypertrophy than by a Mechanical Obstacle to the Circulation*, 205. *Hence the difference in the History of Dropsy in Hypertrophy and in Dilatation*, 206. *Hence also Dyspnœa is comparatively slight and transitory in pure Hypertrophy*, 207. *Hypertrophy with Contraction if extreme causes Obstruction of the Circulation, and its Signs, Dropsy, &c.*

207. Effects Hypertrophy of the left Ventricle on the Brain, 208. *Apoplexy and Palsy more frequent Effects of it than of the Apoplectic Constitution*, 208. *This Constitution generally conjoined with Hypertrophy*, 208. *Hypertrophy a cause of Cerebral Irritations and Inflammatory Action in general*, 209. *Of Opthalmia*, 209. *Of wasting of the Eye*, 209. *Effects of Hypertrophy on the Brain may be partially counteracted by Contraction of the Aortic Orifice—Case*, 210. *A slight Contraction insufficient for this Purpose*, 211. *Effects of Hypertrophy of right Ventricle on the Lungs analogous to that of the left on the Brain*, 211. *Hæmoptysis and Pulmonary Apoplexy the results*, 211.

M. LAENNEC supposes the general symptoms of all organic diseases of the heart to be nearly the same.* It may be said without prejudice to one who has done so much, that, on this subject, both he and all the authors who preceded him, have entertained inaccurate ideas. They had studied these diseases in the aspect under which they most commonly present themselves; namely, complicated one with another; and it is unquestionable that when so viewed, they display a general similarity in their symptoms. But it had never occurred to those authors to analyze each disease in an isolated form. When so examined, although certain symptoms are common to all, they severally manifest differences of a striking kind, obviously dependent on their respective organic peculiarities, and which may, therefore, be fairly regarded as the essential and diagnostic characters of each.

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* De l'Auscult. tom. ii. p. 487.

M. Bertin has the merit of having been the first to display in a clear light the essential pathology of hypertrophy. His distinguished talent for generalization, however, has, I believe it will be allowed, carried him a degree too far. He contends that authors are wrong in having assigned to hypertrophy or *active aneurism* as its symptoms, dyspnœa, suffocation, violet injection of the face, engorgement of the lips and of the venous capillaries in general, passive hæmorrhages, and serous infiltration. He contends that these are the signs not of hypertrophy, but of a coexistent region, viz.—a contracted orifice or any other affection capable of obstructing the circulation; and that pure uncomplicated hypertrophy is characterized by signs of increased activity and energy of the circulation, instead of by dropsy and the other signs of its retardation.

That this is true in reference to the *pure uncomplicated* form of the disease, before embarrassment of the capillary circulation has taken place, will not be denied by any one who has had opportunities of verifying the symptoms by dissection. But M. Bertin is not, in my opinion, supported by sound observation when he says that serous infiltration and the whole class of symptoms bespeaking an obstructed circulation, are totally foreign and repugnant to hypertrophy. The truth I believe to be, that the very same energy of the circulation which gives rise to active hæmorrhages, apoplexy, &c. causes, as its next effect, engorgement of the arterial capillary system; the necessary consequence of which is, serous infiltration and more or less of all the other symptoms indicative of retardation of the blood. The process appears, in fact, to be analogous to that by which serous infiltration is produced in cases of erysipelas, scarlatina, acute rheumatism, &c. I would not be understood by this to mean that capillary congestion is identical with inflammation, but that, as the effects of the two are sometimes the same, we are compelled to admit a close analogy in the mode of their production.

M. Bertin himself unconsciously shows that hypertrophy may produce an obstacle to the circulation, for he says, that when the heart is enormously enlarged the respiration is impeded in a very eminent degree, (d'une manière très notable, Bertin, p. 359). Now, what is the real cause of this impeded state of the respiration? He ascribes it to the encroachment of the heart upon the lungs, but this cause is inadequate: for tumours of a much larger size, as for instance, aneurisms of the aorta, malignant tumours, &c. have existed in the chest, even for years, without producing similar inconvenience.

It is not, therefore, to compression of the lungs that we are to look, as the cause of the dyspnœa and dropsy; but, clearly, to the heart itself; and on reflection it is very conceivable that, when the blood is poured in increased quantity and with unwonted impetuosity into the capillary vessels of the lungs, so as to gorge and obstruct them, the obstruction, *being universal*, must be greater than when a free channel is left open through only a limited portion, as even one half or third, of the organ. For we constantly see, in cases of phthisis, that such a portion is sufficient for maintaining the circulation.

The primary effect of universal obstruction of the lungs by engorgement, is, to produce ædema of their cellular tissue and dyspnœa; whether the latter depends solely on the engorgement or partly also on spasm of the bronchi excited by the irritation of that congestion, is difficult positively to determine, though the latter is highly probable. To this subject I shall revert hereafter. The secondary effect is, to gorge the right side of the heart, and thus impede the return of the venous blood from the system at large; which co-operates with the increased energy of the arterial circulation in producing anasarca.

It must be admitted, however, that hypertrophy does not produce serous infiltration so readily and promptly as a direct, primary obstacle to the return of the venous blood; a fact which admits of a rational and obvious explanation. When there is an obstacle to the return of the venous blood, suppose, for instance, contraction of the tricuspid orifice, two causes conspire to produce the capillary congestion; namely, the direct pressure of the arterial vis-a-tergo, and the retrograde pressure of the retarded venous blood. But when the latter pressure does not exist, when the veins freely receive and transmit their natural proportion of blood, the force of the arterial circulation must be very greatly increased, before it can so far overcome the elasticity of the capillaries as to give rise to engorgement and infiltration.

This satisfactorily accounts for the difference in the history and character of infiltration as resulting, on the one hand, from pure hypertrophy, and, on the other, from contraction of a valve or other primary obstacle to the circulation. In the former case, it appears late, is generally moderate in extent, and requires for its production an aggravated form of hypertrophy; in the latter case, it appears comparatively early, is more copious, and yields with less facility to remedies.

The same reasons that account for the tardy occurrence of dropsy in pure hypertrophy, account, likewise, for another characteristic of this malady when moderate in degree; namely, the slight and transitory nature of the attacks of dyspnœa. For, if the quantity of blood impelled into the lungs by the right ventricle, and the force with which it is impelled, are not very excessive, the pulmonary veins are capable of relieving the engorgement almost as quickly as it takes place, and, consequently, the hurry of the respiration subsides promptly after the removal of its exciting cause.

The sum, then, of all that has been said, is, that pure hypertrophy gives rise to increased force and activity of the circulation, and that, when this force surmounts the natural tonic power of the capillaries, congestion, infiltration, and the other phenomena of an obstructed circulation, ensue.

To these principles an exception presents itself in hypertrophy with contraction, when the cavity of the ventricle is so small as to be incapable of transmitting the natural quantity of blood. In this case, supposing the left ventricle to be the one affected, the arterial circulation sustains a diminution of force and activity; and whether the one ventricle or the other be affected, it creates an obstruction tantamount to that produced by valvular contraction, and, on the same principles, generates dropsy and the other phenomena of a retarded circulation. I have met with three or four cases in which the ventricle was reduced to the size of a small walnut.* Such cases, however, are very rare.

The effects of hypertrophy of the left ventricle on the brain, are so pre-eminently important, that it is necessary to advert particularly to this subject, for the purpose of bringing it prominently into view.

Since the researches of the present day have demonstrated that even a slight thickening of the walls of the heart constitutes a morbid state; and have unfolded to view the connection subsisting between that state and a train of symptoms formerly either wholly overlooked or attributed to other causes; instances of apoplexy supervening upon hypertrophy have been so frequently noticed, that the relation of the two as cause and effect, is one of the best established doctrines of modern pathology. Eight or nine cases of suddenly fatal apoplexy, and numerous cases of palsy, from hypertrophy, have, within a few years, fallen under my own observation. In the majority of them the patient exhibited what is commonly called the "apoplectic constitution;" that is, a robust conformation, a plethoric habit, and a florid complexion: in others these characters were absent; but the total number of the cases of apoplexy from hypertrophy, is much greater than I have witnessed, during the same period, of apoplexy from causes independent of hypertrophy. Whence I am led to believe, with M. M. Richeraud and Bertin, that hypertrophy forms a stronger predisposition to apoplexy than the apoplectic constitution itself; and that, in most instances, those persons who present the apoplectic constitution in conjunction with symptoms of increased determination to the head, are, at the same time, affected with hypertrophy.

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* See one by the writer. Lond. Med. Gaz. Sept. 5, 1829. p. 422.

Nor is it to apoplexy alone, but to cerebral inflammations and irritations of every description, and even to inflammatory action in general, that hypertrophy of the left ventricle gives a tendency. The history of individuals affected with it, frequently presents a striking narrative of violent head-aches, brain fevers, various inflammatory complaints, and states of great nervous irritability and excitation. As the ophthalmic artery is derived from the carotid within the cranium, the eye participates with the brain in the effects of hypertrophy, and is vascular, brilliant, and very prone to ophthalmia. The wasting away of the eye which Professor Testa has remarked as one of the effects of disease of the heart, is, with good reason, supposed by M. Bertin to be connected with ossification of the ophthalmic arteries, a frequent concomitant of hypertrophy of the left ventricle.

The shock of an hypertrophous left ventricle may to a certain extent be intercepted, and its effects on the brain counteracted, by contraction of the aortic orifice. A patient was under the care of Mr. Babington, at St. George's Hospital, Sept. 16, 1829, for a surgical complaint, in whom the walls of the left ventricle were an inch thick, without any change of the cavity; and the aortic and mitral orifices were respectively encircled by a ring of bone as thick as a writing quill. The two valves, though overspread with calcareous scales, were capable of discharging their function. Notwithstanding this extraordinary state of disease, the patient had attained the age of eighty without manifesting symptoms of diseased heart sufficient to arrest his own attention, or that of his medical attendants. His advanced age, indeed, proves that they could not have existed in any considerable degree. In this case, therefore, the valvular contraction appears to have been exactly sufficient to countervail the hypertrophy, and maintain the circulation in a state of equilibrium. The generality of authors, however, have greatly over-rated the power of contraction of the aortic orifice to counteract the effects of hypertrophy on the brain.

They have supposed that a moderate, and even a slight degree of contraction, is sufficient for the purpose. There can be no greater error; and it is one into which they could not have fallen, had they been aware that such a degree of contraction has very little effect in diminishing the strength, tension and regularity of the pulse. To this subject we shall revert in the article on valvular disease.

To have demonstrated the influence of hypertrophy of the left ventricle on the brain, is equivalent to having proved that of the right ventricle on the lungs. For, in the same way that the brain receives directly the shock of the blood which the left ventricle shoots into the aorta, so, the lungs receive *immediately* the impulse communicated to the column of blood, which the right ventricle propels into the pulmonary artery. Consequently, when the walls of this ventricle are augmented in thickness and energy, they impart a corresponding activity to the pulmonary circulation, and sometimes overcome the tonic power of the vessels. (Bertin, p. 352.) Hence ensues hæmorrhage, or what was called by Laennec *pulmonary apoplexy*, from its taking place by the same mechanism as apoplexy properly so called, in cases of hypertrophy of the left ventricle. The hæmoptysis resulting from this cause consists of fluid, red blood, and is generally copious, sudden, and productive of febrile excitement of the circulation. It is, in short, an active, arterial hæmorrhage, and essentially different from that passive species, hereafter to be described, which results from retardation of the blood in the venous capillaries of the lungs.

SECTION V.

SIGNS AND DIAGNOSIS OF HYPERTROPHY.

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The signs of hypertrophy are of two classes: the first, called *general*, consists of its effects on the functions of the economy at large: the second, for which *physical* is the most appropriate designation, comprises the impulse and sounds of the heart and the resonance of the præcordial region on percussion.

According to my experience, neither of these classes of signs, taken separately, is sufficient to indicate disease of the heart with perfect certainty: taken conjointly, they render the diagnosis so easy, that a material error can scarcely be committed.

General signs.—As a systematic arrangement of signs facilitates their registration in the memory, and their employment in the process of catechizing a patient, it may not be irrelevant to state that, in describing those of hypertrophy, I shall follow the course of the circulation; commencing, after having noticed the action of the heart, with the circulation through the lungs, proceeding to that through the aortic system, and concluding with that through the veins.

The description of symptoms which I am about to offer, refers, it must be distinctly understood, to *simple* hypertrophy, when it is not otherwise stated: the symptoms of hypertrophy with dilatation, which will be glanced at incidentally, are only an aggravated degree of the same—as the reader will sufficiently understand, if duly acquainted with the foregoing principles relative to the formation and effects of these diseases. When the dilatation predominates over the hypertrophy, the symptoms, of course, approximate more nearly to those of dilatation.* The symptoms of hypertrophy with contraction will also be noticed incidentally with those of simple hypertrophy.

1. *Palpitation.*—By this is to be understood, a morbidly increased action of the heart both as to strength and frequency. As the hypertrophous heart acts with an energy which, even in its tranquil state, verges on palpitation, and which, under the slightest excitement, actually amounts to it, the patient experiences this symptom more unintermittingly than in any other disease of the organ. It is induced by stimulants of any description: as efforts, particularly that of ascending; mental emotion; flatulence; acidity or bile; spirituous or highly seasoned ingesta, and sometimes by a full meal of any kind. The violence of the attack generally subsides promptly after the operation of

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* Vid. Dilatation.

the exciting cause has been suspended, and little remains but a slight sense of pulsation in the præcordial region. In the advanced stage, however, of hypertrophy, and still more of this conjoined with dilatation, when the circulation has become embarrassed, the paroxysms are sometimes very severe and prolonged, though they never attain that fearful extreme of violence and obstinacy which is witnessed in cases complicated with valvular or aortic obstruction, or adhesion of the pericardium.

2. *Dyspnœa.*—While the enlargement of the heart is moderate, the patient, during a tranquil state of the circulation, feels little or no difficulty of respiration; but he is incapable of making the same corporeal efforts as other persons without losing breath: to use a common phrase, he is "short-winded." After a respite of a few minutes, however, he recovers, and is, therefore, seldom deterred by this symptom from prosecuting his accustomed avocations.

I have frequently observed that an individual who pants on first setting out on a walk, is capable of sustaining great exertions without inconvenience when he gets warm, and the blood is freely determined to the surface.

When the disease has proceeded so far as to occasion dropsy, more or less dyspnœa becomes habitual, and it sometimes occurs, conjoined with palpitation, in paroxysms of excessive severity. From this period, indeed, the symptoms are a compound of those of hypertrophy and those of an obstructed circulation, the latter of which are more particularly considered in the article Dilatation. Hypertrophy with contraction, as already stated, is sometimes accompanied with symptoms of an obstructed circulation.

3. *Cough.*—There is generally little or no cough in the early stages, but it always supervenes when dropsy appears, in connection with which, more or less sanguineous and serous congestion almost invariably takes place in the lungs, and gives rise to the symptom in question. When the hypertrophy is confined to the left ventricle, the cough is milder and later in its appearance than when the right ventricle is affected. I have seen a dry, hacking and wheezing cough amongst the earliest symptoms in young and plethoric females, whom it attacks in paroxysms after any over exertion, as ascending a stair.—It is often also very troublesome on first rising in the morning.

4. *Hæmoptysis.*—This may occur at any period of the disease, and the hæmorrhage, being *active*—the result of a too impetuous discharge of blood into the capillary system—is generally sudden and copious, consists of fluid, arterial blood, and is attended with febrile excitement.

5. *Pulse.*—The pulse in hypertrophy of the left ventricle undergoes, from valvular and other lesions, a variety of modifications which disguise its real nature. It must, therefore, be studied in cases totally exempt from complication. In such, it is almost invariably regular, and bears strict relations in strength and size to the thickness and capacity of the left ventricle. Thus, in simple hypertrophy, it is stronger, fuller and more tense than natural: it swells gradually and powerfully, expands largely, dwells long under the finger and is sometimes accompanied with a thrill or vibration. These characters are still more marked in hypertrophy with dilatation, so long as the hypertrophy is predominant; but when the dilatation has proceeded so far as to diminish the contractile power of the muscular fibres, the pulse, though still full and sustained, is soft and compressible. In hypertrophy with contraction of the cavity, it is strong, hard and tense, but small and cord-like, expanding little under the finger. The action of the carotids corresponds with that of the radials, and they may generally be seen to pulsate from the sternum to the angle of the jaw. In the temporals also a sense of throbbing is usually experienced.

6. *Affections of the head.*—The patient complains of a "rushing of blood to the head" on making any corporeal effort or stooping; of intense

throbbing and lancinating head-aches, aggravated by the recumbent position, and especially by the act either of suddenly lying down or rising up; of vertigo, tinnitus aurium, scintillations and other visual illusions; and sometimes of a lethargic somnolency, which so completely subdues the faculties both of the mind and the body, as utterly to incapacitate him for every species of exertion. These symptoms, if not relieved, terminate in palsy or apoplexy. From this catastrophe the patient is often preserved by the opportune occurrence of epistaxis, to which, happily, he is peculiarly liable. From the circulation in hypertrophy being active in the eye, this organ is bright and sparkling and sometimes vascular or blood-shot.

7. *Complexion.*—The effect of hypertrophy is to heighten the colour so long as the capillary circulation continues unembarrassed, but afterwards to diminish and change it. Every individual, however, does not acquire a florid colour. Whether he acquire it or not, depends, in fact, upon the original complexion, the series of changes being different in those who are naturally florid, and those who are pale. In the former, the colour becomes remarkably vivid, and, being generally accompanied with plethoric turgescence, it gives the aspect of health and good condition. But when the capillary circulation begins to labour, the red

changes into a purplish patch on the cheeks, the nose and lips become more or less purple, violet, or livid, and the intermediate skin becomes sallow and cachectic. In great hypertrophy with dilatation the purple and violet colours are sometimes of the deepest dye. In those who are naturally devoid of colour, hypertrophy either does not excite it at all, or merely increases, in a slight degree, the general vascularity of the face. This vanishes entirely when the capillaries become obstructed, and is superseded by universal cadaverous paleness, extending sometimes even to the lips. They, however, are generally somewhat livid.

8. *Serous infiltration.*—This, for reasons already assigned, seldom appears before the hypertrophy is very considerable or becomes conjoined with dilatation. It frequently shows itself first in the face: a circumstance attributable to the great number and size of the cerebral arteries, and to the force with which the blood is injected into them in consequence of their proximity to the heart. With dropsy supervene, to a greater or less degree, all the other symptoms of an obstructed circulation.

Signs of hypertrophy of the right ventricle.—Hypertrophy of the right ventricle produces, according to Corvisart, a greater difficulty of respiration and a deeper colour of the face than is produced by the same affection in the left ventricle.

Another sign is, the more frequent expectoration of pure, arterial blood.

Turgescence of the external jugular veins accompanied by pulsation synchronous with that of the arteries, was broached by Lancisi as a sign of "aneurism," i. e. hypertrophy with dilatation, of the right ventricle. This sign, though rejected by Corvisart, in my opinion on insufficient grounds, is approved of by Laennec, who found it to exist in every case of rather considerable hypertrophy of the right ventricle, and never in that of the left unless the right was simultaneously affected.* I have rarely known it to be absent in cases where dilatation was conjoined with hypertrophy of the right ventricle. Of such cases, therefore, I regard it as one of the best general signs.

The explanation of the phenomenon offered by M. Bertin appears unsatisfactory. "The jugular or venous pulse," says he, "is seen in those cases only in which dilatation accompanies hypertrophy, and in which the auriculo-ventricular orifice, being greatly enlarged, is no longer completely closed by its valve: thence ensues a regurgitation of blood into the great veins during the contraction of the right ventricle." Hypertrophy with dilatation has certainly, though not always, the effect of enlarging the auriculo-ventricular orifice; but the valve in most instances expands in a corresponding degree; as I have repeatedly found. I apprehend, therefore, that the venous pulsation, in the cases where I have observed it to exist, was not attributable to regurgitation: in substantiation of which opinion I may say, that regurgitation would be attended with a bellows or other such sound: this sound, however, is not found to be a concomitant of jugular pulsation. Is the rationale of the phenomenon as follows? namely, as the ventricle, when hypertrophous, contracts with augmented power, the recoil of the tricuspid valve is preternaturally impetuous: hence, the column of blood in the act of descending into the ventricle, is repelled with such an increase of force, that its impulse is propagated as far back as the jugular veins. This effect will be more considerable when the orifice and valve are enlarged, because the quantity of fluid repelled will be greater. The effect will also be favored by congestion of the great veins, (a state which generally accompanies hypertrophy with dilatation of the right ventricle) because when congested, they are more tense, unyielding tubes and transmit an impulse more readily.

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* Laennec de l'Auscult. tom. ii. p. 505.

But the jugular pulsation is double: a weaker pulsation precedes that occasioned by the ventricular systole. The weaker is occasioned by the auricular systole, and the mechanism of its formation I conceive to be this: at the time that the auricle contracts, the ventricle is in a state of moderate or natural fulness: it therefore offers a certain degree of resistance to the ingress of more blood from the auricle; consequently, so much of the blood compressed by the auricular systole as cannot get forward into the ventricle, is forced back into the veins and causes their pulsation. Some contend that the auricle occasions no jugular pulsation, founding this opinion on the assumption that the ventricle is empty at the moment that the auricle contracts, and that, therefore, the whole of the auricular blood must descend into the ventricle. Such an assumption, however, according to the evidence adduced in the first chapter of this work, is incorrect.

A difficulty has sometimes been experienced in distinguishing jugular pulsation from that of the carotid arteries. Error may easily be avoided by observing that the jugular pulsation is confined to the lower part of the neck, and is far on the humeral side of the carotid. The pulsations of this artery, on the contrary, extend as high as the angle of the jaw, and in the direction of the anterior margin of the sterno-cleido mastoideus muscle.

The jugular turgescence, moreover, disappears in some degree during inspiration and reappears on expiration: which movements, therefore, must not be confounded with the pulsations answering to the systole of the ventricle.

General signs of hypertrophy of the auricles.—There are none that are distinguishable from those of disease or obstruction in the corresponding ventricle or orifice, to which the hypertrophy of the auricles owes its origin. The detection of hypertrophy of the auricles is of little importance, as it is the cause that produced it, which is the source of danger.

Physical Signs of Hypertrophy.

Impulse.—In *simple hypertrophy*, the impulse communicated by the stethoscope while the patient is in a calm state, is usually so strong as distinctly to raise the head of the observer, and sometimes even sufficient to produce a shock disagreeable to the ear. The greater the hypertrophy, the longer this heaving takes for its performance. When the malady exists in a great degree, we evidently perceive that the heaving takes place with a gradual progression; it seems as though the heart swelled and applied itself to the parietes of the chest, at first by a single point, then by its whole surface, and finally sank back in a sudden manner. This sinking back, which I have been in the habit of designating by the term *back-stroke*, is occasioned by the diastole of the ventricles, during which action the heart sinks back from the walls of the chest with a force greater in proportion to its thickness and capacity. Accordingly, the *back-stroke* is strongest in hypertrophy with dilatation, but it may also be very considerable in simple hypertrophy. In the healthy heart it is not perceptible; neither is it in dilatation without hypertrophy.

A strong, slowly heaving impulse, then, is the principal sign of simple hypertrophy; and the affection may be known to be greater when the impulse is followed by a back-stroke. Both these signs exist in hypertrophy with contraction, but in a less degree, and the back-stroke may be absent if the disease is not great.

In simple hypertrophy and that with contraction the impulse is seldom perceptible much beyond the præcordial region, except during attacks of palpitation.

In hypertrophy with dilatation the signs are a compound of those of hypertrophy and those of dilatation. The contraction of the ventricles can easily be felt by the hand applied to the præcordial region, and we find, especially during palpitation, smart, violent shocks, which strongly repel the hand. If we attentively examine the patient when most calm, we see that his head, his limbs and even the bedclothes, are strongly shaken at each contraction of the heart. The pulsations of the carotids, the radials, and the other superficial arteries are often visible. The impulse of the heart can sometimes be distinctly felt under the clavicles and on the left side of the thorax; sometimes even in the back, especially in meagre subjects and children.

In *hypertrophy with a predominance of dilatation*, the impulse is ordinarily not considerable; but it becomes very marked during palpitation, especially if accompanied with fever, and it has a very different character from that occasioned by simple hypertrophy. The beats are strong, hard, and produce a shock analogous to the blow of a hammer; but the blow seems to strike a small space, it expends itself as it were on the thoracic parietes and does not communicate to the head a heaving proportioned to its force: it differs, in short, from the impulse occasioned by great hypertrophy, in the circumstance that, in the latter, the ventricles in a distended state, seem to heave with their whole length against the thoracic parietes, which yield to the effort; while, in the former case, the point only of the heart seems to strike the parietes with a sharp, smart, accurately circumscribed blow, only capable of producing a sort of concussion rather than a real heaving. The same species of impulse takes place in purely nervous palpitations, where I have called it jerking.

When the impulse is increased on one side only of the præcordial region, that is, under the inferior part of the sternum, for the right side, and between the cartilages of the fifth and seventh left ribs, for the left, we infer that the corresponding ventricle only is affected: and when it is increased on both sides, we conclude that both are affected, which is the more common case.

In hypertrophy, and hypertrophy with dilatation, free from valvular disease, the beats of the heart, even during palpitation, are rarely irregular, unless when they become enfeebled by excessive dyspnœa or by failure of the vital powers on the approach of dissolution.

The impulse of the heart is diminished by loss of blood, diarrhœa, any exhausting disease, rigid and long continued abstinence, and, in general, by all the causes capable of producing debility. Consequently, a moderate hypertrophy might, without due care, be overlooked in a patient under any of these circumstances.

The impulse of the heart, moreover, occasionally ceases entirely (even in cases of very marked hypertrophy) when there supervenes intense dyspnœa referable to some affection of the lungs, especially peripneumony, pleurisy, œdema of the lungs, asthma, and the congestions which form during the last moments of life. The sounds likewise diminish or even entirely cease: no inferences, therefore, should be drawn from an exploration made under such circumstances.

Sounds.—Hypertrophy has the effect of deadening the sounds of the heart. In *simple hypertrophy*, the *first* sound, i. e. that produced by the ventricular contraction, is duller and more prolonged than natural, in proportion as the hypertrophy is more considerable; and it generally terminates without any interval in the second sound. When the hypertrophy exists in an extreme degree, the first sound becomes nearly, and sometimes wholly extinct. The *second* sound, i. e. that produced by the ventricular diastole, is very brief and dull; in extreme cases, it is scarcely perceptible. The interval of repose is shorter than natural in consequence of the first sound being longer. Both sounds are proportionably weaker, when the ventricle is contracted as well as hypertrophous. In most cases the sounds can scarcely be heard under the left clavicle and at the upper part of the sternum. Sometimes they cannot be heard farther than the impulse can be felt, that is to say, scarcely beyond the limits of the præcordial region.

Each sound of the heart, though essentially one, consists of the sounds of the two sides united. This is proved by a bellows murmur in the left præcordial region being audible in the right, and vice versâ. It does not follow, therefore, that because one ventricle is hypertrophous, the sound of the heart in general should be very limited in its range; for that of the other will be heard over an extent proportioned to its intensity, though not quite so far as when strengthened by its fellow. On the other hand, a morbidly increased sound of one ventricle, as by dilatation or a bellows-murmur, will be heard *alone* at points beyond the range of the natural sound of the other or healthy ventricle. Accordingly, it is only in hypertrophy of both ventricles that we must expect to find the sounds confined within very narrow limits.

I have not been able to verify the remark of Laennec that, "in hypertrophy, often when a strong heaving without any first sound, is felt in the præcordial region, and the second sound can scarcely be distinguished, the latter is heard alone under the clavicles and even on the back; and, in less severe cases of this kind, it is always heard more distinctly in these places than in the præcordial region, especially in meagre and narrow-chested persons." I cannot understand on what principle it could be thus, even supposing the auricular contraction were, as he imagines, the cause of the second sound. For, as the right auricle and ventricle are both in immediate proximity and partly in contact with the sternum, it is inconceivable how the sound of either should be less distinct not an inch from its source, than at a remote point. I have, however, found Laennec's remark true when the sound was drowned in the præcordial region by a bellows-murmur or pulmonary râle: the explanation of which is, that the second sound, being of a more acute nature than the murmurs, is more readily transmitted to a distance.

On the same principle also, is to be explained another doctrine of Laennec, the accuracy of which I have frequently verified: namely, that "in healthy subjects, but in whom the heart has rather thin walls, the second sound is sometimes stronger under the clavicles than the first, although the same difference is not observable in the præcordial region." The reason of this is, that the second sound, being more acute, is more easily propagated.

In *hypertrophy with dilatation* the sounds are increased. The first is, as it were, a compound of the sound of dilatation and that of hypertrophy: namely, from dilatation it derives a loud, abrupt or flapping commencement, and from hypertrophy, a prolonged termination like a respiratory murmur. The second sound, though not in general changed in its character, is louder than natural. These sounds may frequently be heard over the whole chest both posteriorly and anteriorly, especially in children and meagre subjects.

In hypertrophy with dilatation the sound of the ventricular contraction is sometimes accompanied with bellows-murmur. This I have found to be almost always the case when the heart is extremely large, and contracting with vehemence. (Vid. Causes of Murmurs, p. 63.) The sounds of the heart in every form of hypertrophy may be diminished by the same causes that diminish the impulse.

Resonance[*] of the præcordial region on percussion is deficient in simple hypertrophy if the heart is considerably enlarged; but, as hypertrophy and dilatation is the disease in which the organ attains the greatest volume, it is that in which resonance is most frequently, and most extensively deficient. In all cases of considerable enlargement, the dulness as well as the impulse, are lower down than natural except in adhesion of the pericardium; for the heart is then more or less braced up by the adhesion.

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[*] See much valuable information on this subject in the "Procédé Opératoire" of Mr. Piorri. Paris, 1830. p. 112, et seq.

SECTION VI.

PROGRESS, TERMINATIONS AND PROGNOSIS OF HYPERTROPHY.

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Progress and termination of hypertrophy.—Hypertrophy, while moderate and not complicated with any mechanical impediment to the circulation, is productive of very little inconvenience. This is especially true with respect to children. In them, the heart is naturally larger in proportion than in adults; and in many this amounts to a very considerable degree of hypertrophy with dilatation, accompanied with greatly increased impulse and sound; yet the general symptoms manifested by such are often scarcely appreciable, and the increased action itself subsides towards the period of puberty by the establishment of a more correct proportion and equilibrium between the heart and the system.

At the adult age also, and during the whole period of manhood, an individual of an otherwise sound and vigorous constitution may be affected with hypertrophy to a moderate extent, without experiencing any sensible deterioration of the general health, (with the exception of being more liable than others to phlogistic and cerebral affections,) or any diminution of muscular force and activity; and if his habits with respect to diet and exercise be moderate, he may pass a long series of years, and even attain the extreme period of senility, without being conscious that he is the subject of organic disease. The only general signs denoting the existence of the malady, will be, perhaps, a little shortness of breath on exertion, and occasional feelings of slight palpitation. Amongst the labouring classes these symptoms, even in a considerable degree, are so little regarded, that their presence is often disavowed by the patient, though manifest to the physician. I recently saw an athletic, hard-working man, weighing, according to his own account, not less than twenty stone, with enormous hypertrophy and dilatation, who assured me that "his palpitation had quite left him for a month," yet the heart was acting with a violence that was truly astonishing.

If, however, an individual affected with hypertrophy abandon himself to intemperate living, or engage in occupations requiring great corporeal exertion, he rarely fails to bring on either apoplexy, palsy, hæmoptysis, or an irremediably aggravated state of the disease which embitters the remainder of his existence, as well as curtails its span.

The celerity with which these accidents are induced, depends on circumstances. In general, the progress of hypertrophy is very slow and gradual, but in some cases it is rapid: in several instances I have known it terminate fatally within a year from its commencement.

The circumstances occasioning these variations are connected with, 1. the form of the disease; 2. its complications; 3. the nature and intensity of the external exciting causes; and, 4. the constitution of the patient.

It is of the utmost importance that the practitioner be able to form some estimate of the influence of these circumstances; for it is by this means only that he can foresee the course of the disease, and direct his treatment with judgment and decision. It may be useful, therefore, to enlarge a little on this subject.

1. The progress and termination of hypertrophy

are influenced by the form of the disease. *Simple hypertrophy* is more apt than any other form to induce apoplexy while the patient is apparently in perfect health. This is to be accounted for by its tendency to create plethora, while, at the same time, it does not incapacitate the patient for active corporeal exercise, and the pleasures of the table. If a premature death does not occur from apoplexy or hæmoptysis, simple hypertrophy runs a more chronic course than any other form of the disease.

Hypertrophy with dilatation, especially if great, is a far more harassing, dangerous, and, if I may be allowed the term, *acute* affection than the preceding. All its symptoms are more violent, and its course is more rapid. It is somewhat less apt to produce unexpected attacks of apoplexy; probably because the greater dyspnœa which it occasions deters the patient from violent exercise and high living. When once the palpitation and dyspnœa have attained such an extent as imperatively to demand periodical bleedings at brief intervals, the malady hurries with an uninterrupted course to its fatal termination.

2. The progress and termination of hypertrophy are influenced by its complications. When hypertrophy is connected with contraction of an orifice or any other obstacle to the course of the blood, the symptoms are greatly aggravated. For, in the

first place, in consequence of that obstacle, the hypertrophy proceeds to a greater extent; and, secondly, the violent struggles of the heart to surmount the obstacle, subvert the general balance of the circulation. To speak more explicitly, suppose the obstacle to be situated in the aortic orifice. While the left ventricle is palpitating to disgorge itself through the contracted aperture, the right, acting in concert with it, deluges the lungs with an inordinate quantity of blood; whence ensues a paroxysm of dyspnœa: next, in consequence of the pressure of blood through the lungs, the supply to the left ventricle is increased: this ventricle, therefore, instead of relieving its engorgement by palpitation, only aggravates it, and the fit does not subside until either the heart becomes gradually exhausted by its own efforts, or (what is more common) until the internal congestion is relieved by determination to the surface, or a copious discharge of watery mucus from the lungs. The most violent paroxysms of palpitation and dyspnœa that I have witnessed, have occurred in the particular complication described. In others, however, there may exist a greater feeling of suffocation, as will hereafter be explained in the chapters on diseases of the valves, and on polypi.

Adhesion of the pericardium, which rarely fails to produce hypertrophy with dilatation, is an extremely formidable complication of this malady. It greatly aggravates all the symptoms and accelerates the fatal event. It is not unusual for this to take place within the period of a year, and I have known it occur in nine months.

Febrile or inflammatory complaints supervening upon an advanced degree of hypertrophy, exasperate the malady in a surprising manner, so as not unfrequently to carry off the patient in the course of a few days. The effect seems to be produced by the febrile excitement keeping up, as it were, a perpetual fit of palpitation and embarrassment of the circulation, which the constitution cannot support beyond a brief period. Peripneumony has pre-eminently this effect: apparently because it not only excites the heart, but obstructs the circulation through the lungs.

3. The progress and termination of hypertrophy are influenced by the nature and intensity of the external exciting causes.

The principal of these are, over-exertion, excesses at table, and mental perturbation, the latter of which, though not strictly external, may be ranged under this head. The effect of these requires no explanation; but it may be said that the injurious influence of over-eating is greatest in simple hypertrophy, because it generates plethora and increases the tendency to apoplexy; while over-exercise and intemperance are more prejudicial in hypertrophy with dilatation, because they increase the dilatation, which is the more dangerous part of the disease.

4. The progress and termination of hypertrophy are influenced in a remarkable degree by the constitution of the patient. The robust resist its encroachments much longer than those who are delicate and effeminate: and if the former, either from bad air and want of exercise, from disease, or from age, become unhealthy, emaciated and feeble, they are rendered much more susceptible of the effects of the disease. This (if I may indulge in a mere speculation) is possibly in consequence of emaciation taking place to a greater extent in the muscular and adipose tissues, than in the internal viscera, whence, the latter becoming predominant in size, the equilibrium between the heart and the system is subverted. This is exactly the converse of what occurs at the period of puberty in those who had laboured under enlargement of the heart when children; for, in the latter, the equilibrium is restored by the system enlarging in proportion to the size of the heart.

Prognosis.—The general prognosis is favourable in the early, and unfavourable in the advanced stages of the disease. The particular prognosis must be formed on an estimate of the various circumstances of the case, formed according to the above rules.

SECTION VII.

TREATMENT OF HYPERTROPHY.

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As it is easier to diminish the nutrition of the heart than to increase it, or to remove a valvular or other mechanical obstruction, it is very conceivable that hypertrophy is more susceptible of cure than any other organic affection of the heart. Before the introduction of auscultation, when practitioners could not distinguish disease of the heart with any certainty, and seldom before it was in an advanced stage, they generally considered it as hopeless, and contented themselves with palliating urgent symptoms. Nor can this be a subject of surprise, for in that stage the disease most frequently *is* hopeless so far as a cure is concerned. But, since, by the aid of auscultation and the improved knowledge of general symptoms to which it has led, it has become possible not only to detect the slighter degrees of hypertrophy or dilatation, but even the mere tendency to those affections; and since it has been fully proved that, in their early stages and sometimes even when far advanced, they are within the resources of the curative art, the practitioner would be wanting in the performance of his duty to his patient were he not to aim at effecting a cure, rather than content himself with merely palliating symptoms.

In the treatment, it is obvious that the first care should be, to remove any known exciting cause of the malady. It is equally obvious that as this malady consists in an increased power and action of the heart, blood-letting and other reducing and tranquillizing means are the appropriate remedies. Laennec strongly recommends that they be employed with courage and perseverance, on the plan of Albertini and Valsalva. I cannot say that my own observation leads me to coincide entirely in this opinion. I shall first, therefore, give a sketch of the treatment alluded to, the sanction accorded to which by names of the highest authority renders it at least deserving of the most attentive consideration; and shall afterwards point out in what respects it appears to me to be objectionable.

This treatment, according to M. Laennec, ought to be prosecuted in an energetic manner, especially at the commencement; and, in aiming to enfeeble the patient, we ought much more to fear resting short of the mark, than exceeding it. We should commence by abstracting blood as copiously as the patient can support without falling into a state of sinking, and we should repeat the operation every two, four, or eight days, until the palpitation has ceased, and the heart no longer gives, under the stethoscope, more than a moderate impulse. We should, at the same time, reduce to at least one half, the quantity of aliments which the patient ordinarily takes, and diminish even this quantity, if he preserve more muscular strength than suffices to take, step by step, a walk of a few minutes in the garden. In a stout adult, Laennec usually reduces the quantity to fourteen ounces a day, amongst which he thinks there should be only two ounces of white animal food. If the patient wish to take broth or milk, he counts four ounces of these liquids for one of animal food. Wine ought to be interdicted. When the patient has been about two months without experiencing palpitation, and without strong impulse of the heart, we may dispense with the bleedings, and somewhat diminish the severity of the regimen if habit has not yet been able in any degree to reconcile him to it. But it is necessary to revert to the same means, and with equal rigour, if in the sequel the impulse of the heart increase again. We ought not to have confidence in the cure until the expiration of a year of complete absence of all the symptoms, and especially of all the physical signs, of hypertrophy. We must be afraid of allowing ourselves to be deceived by the perfect calm which blood-letting and abstinence sometimes very promptly produce, especially if we have commenced the treatment at a period when the hypertrophy was already accompanied with extreme dyspnœa, with anasarca, and with other symptoms which gave reason to fear an approaching death.

If we begin the treatment of hypertrophy of the heart at a period when it has already produced severe effects, particularly anasarca, ascites, œdema of the lungs, and a very marked state of cachexy, we ought not on that account to shrink from bleeding and abstinence.

To obtain success by the treatment described, it is necessary, according to the same author, that the physician and the patient arm themselves with almost equal patience and firmness; for it is not more difficult for the latter to resign himself to a perpetual fast and frequent blood-lettings, than for the former to struggle daily against the opposition of relations, friends, and the discouragements which cannot fail to seize upon the patient in a treatment which ought to continue at least several months, and sometimes to be prolonged during several consecutive years.

Such is the manner in which M. Laennec employs the treatment of Albertini and Valsalva, and he states that he could cite a dozen instances of cures of hypertrophy, either simple or with dilatation, which have not been falsified for several years. One important case, which he details, seems to prove that the treatment causes atrophy of the heart; for the organ was remarkably less than the fist of the subject and was shrivelled or wrinkled in a longitudinal direction.

My objections to the treatment described are founded on the circumstance that, though I have invariably found the greatest benefit to be derived from sparing abstractions of blood at intervals of two or three weeks or more, I have constantly noticed that when, from the severity of the dyspnœa and palpitation in the advanced stages of the complaint, the practitioner was induced, or thought himself compelled, to resort to frequent bleedings at short intervals, the patient, though perhaps temporarily relieved, progressively declined from that moment, the paroxysms recurring more frequently and with greater violence, until they eventually terminated in his destruction. Now, on comparing a patient under these circumstances with one under the influence of mere reaction from loss of blood, the analogy appears to me to be very intimate. In both, the violence of the heart's action, so far from being repressed by a reiteration of the blood-letting, is only increased: in both the blood is, and necessarily must be, attenuated and deteriorated in consequence of the fibrinous portion and red globules being replaced to a greater extent than natural by serum, which is more expeditiously regenerated.

These, then, are apparently the causes of the patient's decline. The prejudicial effects of the reaction are sufficiently obvious; while the state of the blood not only contributes in all probability to the reaction, as explained p. 75, but, by its deteriorated quality, is unsuitable for the due nutrition and conservation of the system.*

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* It does not follow that, though the treatment of Albertini and Valsalva be unsuitable for hypertrophy, it is equally so for aneurism of the aorta; as in the latter the object is, to produce a sudden and extreme, though temporary reduction of the force of the circulation, in order to promote the formation of fibrinous coagula in the sac:—an expedient which is sometimes perfectly successful in aneurism, but which will not have the same effect on hypertrophy.

It would appear, therefore, that the indications in the treatment of hypertrophy, are, to diminish the quantity, without materially deteriorating the quality of the blood; and to do this in such a manner as, without producing reaction, permanently to enfeeble the action of the heart and the energy of the circulation. These indications have seemed to me to be fulfilled in the safest and most effectual manner by the following means.

Four, six or eight ounces of blood should be taken every two, three, four or six weeks according to the strength of the patient, and sufficient to keep down palpitation, dyspnœa, and strong impulse of the heart. If the head be much affected the blood should be drawn by cupping from the nape of the neck. The diet should consist of white animal food and liquids in small quantity, unless, from the advanced state of the disease, the blood be so impoverished as to be insufficient for the maintenance of the system; when a more nutritious, though still spare diet may be allowed. Every thing stimulating, as spirituous and fermented liquors and highly seasoned dishes, should be avoided. Any exercise taken, should be so gentle as never to hurry, and, if possible, never even in the slightest degree to accelerate the circulation. When the action of the heart appears to increase, and yet general bleeding is not expedient, three or four copious and watery alvine evacuations should be procured daily by saline aperients, of which none answer better than one or two drachms of sulphate of magnesia in infusion of roses twice or thrice a day. This may be continued for a week or ten days according to the effect; and either the same or some analogous aperients should be employed habitually in sufficient doses to keep the body gently open and to procure, if possible, liquid evacuations. When salines are used habitually, their debilitating effects on the intestinal canal may be in a great measure counteracted by adding to the infusion of roses an equal quantity of Comp. Infus. of orange-peel and six or eight minims of dilute sulphuric acid.

In addition to purgatives I have seen the most decided advantage result from diuretics, and not only when there was dropsy, but also when there was none. Their mode of operation appears to be ultimately the same as that of purgatives: namely, they drain off the serous portion of the blood. I have found many patients, conscious of the benefit which they derived from this class of remedies, in the constant habit of taking cream of tartar, broom-tea and other similar popular medicines. One patient affected with contraction of the mitral valve to the size of an ordinary pea, by these means warded off dropsy, beyond the slightest œdema of the feet, for ten years.

When decided dropsy appears, it must be combatted by the most efficient diuretics—the supertartrate, tartrate, acetate and nitrate of potass, squill, digitalis, spirit of nitric æther, decoction of broom, &c. with mercury if not contra-indicated. As no class of remedies is more variable and uncertain than this, when one fails another should be resorted to; and it not unfrequently happens that a weaker is more successful than a stronger. Should diuretics wholly fail, hydrogogue purgatives, as elaterium, tincture of jalap, infusion of senna with tartrate of potass, &c. are often invaluable substitutes.

The state of the stomach and of the biliary secretion, should never be overlooked in hypertrophy, as their derangements are amongst the most efficient exciting causes of palpitation. The remedies suitable for dyspepsia and bile are therefore to be resorted to. I deem it unnecessary here to enlarge on them, and on the treatment of dropsy, cough, dyspnœa &c., as these subjects will be found fully discussed in the chapter on disease of the valves.

It frequently happens that, notwithstanding the most judicious use of the remedies mentioned, the irritability of the nervous system frustrates their tendency to reduce and tranquillize the action of the heart. In this case sedatives are eminently useful, and the best effects often result from tincture of digitalis to the extent of m. xx or xxx twice or thrice a day; from two or three drops of hydrocyanic acid administered as often; from three or four or more of extr. of hyoscyamus or conium once or twice a day, and from acetate of morphia.

The above, and indeed every other mode of treatment is unavailing if not *steadily* pursued, and it must be pursued for one, two, three, or more years. Thus employed, I have found it effect cures in a considerable number of instances, some of which were advanced even to the second degree. In the first degree, especially before the period of puberty, this fortunate event is often obtained although bleeding be resorted to only at long intervals, as from six weeks to three months.

CHAPTER II.

DILATATION OF THE HEART.

SECTION I.

ANATOMICAL CHARACTERS WITH CLASSIFICATION AND NOMENCLATURE OF DILATATION.

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THE disease commonly termed dilatation of the heart, consists in an amplification of one or more of its cavities.

Although I have seen the muscular substance healthy in every form and degree of this affection, in general it is not so. For, when the dilatation is great, and the parietes are feeble in proportion to the quantity of blood which they have to propel, the muscle is usually more or less softened and flaccid, and in some cases of a deeper red, in others paler or more fawn-coloured, than natural. (Gillan, Anderson, Mrs. —l—n). The deep red dye is attributable to venous engorgement of the muscular substance, resulting from stagnation of the blood within the heart. The softening is sometimes so great that the substance readily breaks up under the pressure of the fingers.

Dilatation occurs with three different states of the ventricular parietes as to thickness: namely, the thickened, the natural, and the attenuated states. It accordingly resolves itself into three natural varieties corresponding with these states.

1. *Dilatation with thickening*, in which the cavity is enlarged and the walls thickened.

2. *Simple dilatation*, in which the cavity is enlarged and the walls of their natural thickness.

3. *Dilatation with attenuation*, in which the cavity is enlarged and the walls attenuated.

The first variety is identical with that variety of hypertrophy called *hypertrophy with dilatation*, but different names are employed for the two, in order to express, in the former, a predominance of dilatation, and in the latter, of hypertrophy. The second variety is perfectly identical with *hyper-*

trophy by increased extent, with natural thickness, of the walls; but it is better to employ the term *simple dilatation* when the dilatation is so great that its symptoms predominate over those of hypertrophy.

Two, or all three of the forms of dilatation are sometimes found together, in different parts of the same cavity. It is sufficient to notice the fact, without perplexing the memory with a distinct appellation for cases of this compound nature.

The anatomical characters of *simple dilatation* and that with *thickening* are described in the chapter on hypertrophy. To *dilatation with attenuation* we now direct our attention. It seldom affects one ventricle without the other. The emaciation may proceed to such an extent as to reduce the most substantial part of the left ventricle to two lines in thickness, and the apex, to a mere membrane. In a case lately under my observation the prevailing thickness was two lines, (Lambert) and a portion of the apex consisted solely of the internal and external membranes, strengthened by a deposition of lymph on the outside. Extreme attenuation is more common in the right, than in the left ventricle. In either, the columnæ carneæ appear stretched and spread. The inter-ventricular septum is proportionably much less attenuated and softened than the other parts. Dilatation takes place more in the transverse, than in the longitudinal direction of the ventricles, and it accordingly communicates to the heart an unusually spherical form, so that the diameter of the organ near the apex is almost as wide as at the base, the apex itself being often scarcely distinguishable. This alteration of shape is the best criterion for determining whether a heart is dilated or not when the enlargement is so inconsiderable as to render the question doubtful.

When both the auricle and ventricle are much dilated, it is not unusual to find the intermediate aperture widened and its valve sometimes not large enough to close it.

Laennec, although he had never seen a case of rupture of the heart from dilatation, believes with Burns, that it may occur; particularly as dilatation is generally attended with softening. I witnessed a case of this kind, a few years ago. The patient, who was aged upwards of seventy, fell back suddenly while on the night-chair, and immediately expired. A fissure an inch in length was found in the left ventricle, its substance was softened and of a deep violet colour, and the cavity of the pericardium was gorged with blood. Dr. Williams communicated to me the case of a relation of his who died from rupture of the heart in a somewhat similar way; but the orifice through which the blood escaped was small and round, surrounded by dark ecchymosis. The patient (a lady of fifty-eight) had been subject to severe angina for some months before her death. He suspects that there was both attenuation and softening in this instance.

In order to judge accurately of dilatation of the auricles, it is necessary to have distinct ideas respecting their natural form and dimensions. The four cavities of the heart are very nearly equal in capacity; but, as the parietes of the auricles are very thin, and those of the ventricles are thick, the auricles, when simply full and not distended, form only about one-third of the total volume of the organ; or, what is the same thing, the volume of the auricles equals about half that of the ventricles.* The right auricle, being generally found in a state of distention, and being of a more elongated, flattened form than the left, has the appearance of being considerably larger, though in reality it is only a little so.

Distention, taking place during the last moments of life, and observable, though more rarely, in the left auricle as well as in the right, constitutes the great source of fallacy in determining after death whether these cavities are really dilated or not;

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* Laennec de l'Auscult. tom. ii. p. 523.

for the engorgement, though only of a few hours' duration, may stretch them to a magnitude almost equalling that of the ventricles.

M. Laennec has given good criteria, by which a dilated, may be distinguished from a distended auricle. An auricle simply distended is tense, and through its thinnest parts distinctly shows the dark blood within. One dilated does not present the same appearance of tension, and its parietes are more opake. When the blood is evacuated through the vessels without cutting into the cavities, the latter, if merely distended, return at once to nearly their natural size: whereas, if dilated, they maintain almost the same size which they had when full. Dilatation of the auricles, as already stated, scarcely ever exists without more or less thickening of their parietes.

The method of distinguishing distention from dilatation is much the same in the ventricles as in the auricles: namely, when merely distended, they are found enlarged, firm, and tense; but these conditions almost entirely disappear, when the blood is pressed out through the natural apertures. On the contrary, when truly dilated, they have no appearance of tension, are more or less flaccid, and the enlargement persists after the blood has been evacuated.

SECTION II.

MODE OF FORMATION, WITH THE PREDISPOSING AND EXCITING CAUSES OF DILATATION.

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DILATATION of the heart is a purely mechanical effect of over-distention. Blood, accumulated within its cavities, exerts a pressure from the centre towards the circumference, in every direction; and when once it surmounts the resistance offered by the contractile and elastic power of the parietes, these necessarily yield and undergo dilatation. The rapidity with which this process takes place, and the extent to which it is carried, depend on the degree in which the distending, exceeds the resisting force: and as the latter bears a direct ratio to the volume of the muscle, supposing it to be healthy, it follows that those cavities which have the thinnest parietes, are, cæteris paribus, the most susceptible of dilatation.

Accordingly, we find that the right ventricle is more frequently and promptly dilated than the left, and the auricles, than either.

In order to produce permanent dilatation, the operation of the exciting cause must either be prolonged for a certain time, or frequently repeated at brief intervals. Contraction of an orifice, for instance, acts in the former manner; and nervous palpitations, and occupations requiring constantly renewed muscular efforts, produce their effect in the latter way. When the operation of the cause is only brief and transitory, the result is merely a temporary *distention*, from which the muscle recovers itself by its own elastic and contractile reaction so soon as the distending force is removed. This cannot be regarded as a pathological state, and it must, therefore, be carefully distinguished from genuine dilatation.

The exciting causes of dilatation, are, 1st. deficient power of the heart, whether congenital or acquired, in proportion to the system: 2d. in general terms, all obstructions to the circulation, whether situated in the orifices of the heart, or in the aortic, or pulmonary system. The latter class of causes are, in fact, essentially the same as the exciting causes of hypertrophy. For, as stated under hypertrophy, it depends on the proportion which the resistance of the muscle bears to the distending force, whether the one affection or the other be produced. When, therefore, dilatation occurs in one of the cavities with naturally thick walls, in which we should more properly expect hypertrophy, it must be ascribed, either to a congenital disproportion of the heart, in consequence of which the cavity in question is thinner, and therefore more disposed to dilatation, than natural; or it must be attributed to the obstruction, from its nature or situation, bearing more in proportion on that particular cavity, than on any other. It is from overlooking these considerations, respecting the relations of the resisting and distending forces to each other, that some have excluded dilatation from the catalogue of mechanical diseases, and supposed that it takes its rise in any cavity of the heart either by chance, or by some vital predilection, some vague, unintelligible predisposition.

Dilatation occasionally affects only a single ventricle, and it is generally the right; but much more commonly it attacks both. The auricles, being protected by their valves from the direct influence of the numerous causes of pressure which operate on the ventricles, are far more exempt both from dilatation and hypertrophy. But when the auricular valves are diseased, whether their state be that of contraction, which impedes the transmission of the auricular blood, or of permanent patescence, which allows a regurgitation of the ventricular, the auricles, suffering unnatural distention, become dilated.

It is seldom that dilatation of the auricles occurs under any other circumstances than those of disease of their valves: so seldom, indeed, that Laennec does not recollect to have seen an instance, though he does not deny the possibility of the occurrence. More instances than one, however, have fallen under my own observation, and I have generally found the dilatation connected with some circumstances, which rendered the ventricle incapable of freely evacuating its contents. It is natural, indeed, to suppose, that when such is the case, the stagnation of blood in the ventricles must, for the time, have an effect in distending the auricle equivalent to that produced by contraction of the auriculo-ventricular valve; and, considering the frequency of stagnation in the right ventricle, we might at first expect dilatation of the corresponding auricle from this cause, to be frequent. But it must be remembered that, for the production of the disease, it is necessary that the operation of the cause be permanent, or at least very prolonged. Such, however, is seldom the case with the stagnation in question; for a ventricle, though so feeble in itself, or so encumbered by an obstacle before it in the course of the circulation, as to become gorged during an accelerated state of the heart's action, will, when tranquillity is restored, transmit its contents with a facility that could scarcely be anticipated. During such intervals, therefore, the muscular fibres of the auricle recover their contractile power, and restore the cavity to its natural size.

SECTION III.

PATHOLOGICAL EFFECTS OF DILATATION AND MODE OF THEIR PRODUCTION.

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" AUTHORS," says Bertin, " have entered into long disquisitions on what are called the general symptoms of dilatation or aneurism of the heart, and they have allowed themselves to fall into great errors, in considering dilatation a *primitive* malady instead of regarding it as consecutive to another lesion, which was the source of the symptoms that they attributed to the dilatation itself. I have already proved," continues he, "that this malady necessarily supposes an obstacle to the course of the blood; but this obstacle, at the same time that it gives rise to an aneurism of the heart, produces other striking phenomena, such as engorgement of the vessels, serous infiltration, passive hæmorrhages, &c. These phenomena have been taken for the effects of the dilatation of the heart, while this has, strictly speaking, no other relation with them than as being a result of the same cause, that is to say, of an embarrassed state of the circulation."

I cannot concur with M. Bertin in these opinions. It is true that in order to produce dilatation, there must exist a weight or pressure of the circulation upon the heart, greater than the organ is capable of sustaining: and it is true that such pressure may be occasioned by the mechanical obstacles to which M. Bertin ascribes it: namely, contraction of the orifices of the heart, diseases of the aorta, and all maladies which impede the course of the blood, whether in the lungs, or in the system of the great circulation.* But it is equally true that the same pressure on the heart may result, not from increased weight of the circulation, but from deficient power of the heart; and such is its cause in those, who, by original conformation, have the organ thin, in proportion to the size of the body. I believe that this is a more powerful and certain cause of dilatation than the impediments alluded to by Bertin, for the malady prevails principally in the female sex, in whom the walls of the heart are, in general, thinner than in men, while, at the same time, women are less exposed than men to the exciting causes of dilatation, as they lead a more tranquil temperate life, and are less subject to diseases of the arteries and valves. Another class in whom debility of the heart exists as a cause of dilatation, comprises those who have had the organ softened or otherwise enfeebled by disease: an effect not unfrequently produced by typhoid fever, and by inflammation of the substance and membranes of the heart.

Dilatation, then, occurring under the circumstances described, is as justly entitled to the rank of a *primitive* disease as hypertrophy: for as, in both, the disease depends, not on the pressure of the circulation, but on the manner in which the heart resists that pressure; in both, the organ itself is the part where the disease originates: the only difference being, that the effect is produced in the one case by deficient, and in the other, by superabundant power of the muscle.

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* Bertin, p. 380.

In the next place, M. Bertin has, in my opinion, attributed far too much to the lesion of which he considers dilatation to be the effect, when he says that this lesion is the sole cause of all the symptoms which authors have been in the habit of ascribing to dilatation. It is true that when the lesion is so great as to constitute an extreme obstacle to the circulation, it may produce the symptoms in question; but it does not produce them, or only in a very slight degree, when the obstacle is not extreme. I have repeatedly witnessed cases in which a well marked, if not a considerable obstacle, as a contracted valve or a dilatation or aneurism of the aorta, had subsisted for a long period, even for years, without producing any material symptoms of an obstructed circulation; but the moment that dilatation of the heart supervened, the symptoms made their appearance in an aggravated form. I apprehend, therefore, that the heart is the part mainly concerned in their production: nor do I think this opinion less tenable because the symptoms are more severe when enlargement of the heart co-exists with an obstacle, than when the enlargement exists alone; for it is natural to suppose that when two causes conspire to produce the same effect, that effect should be greater. But this is not all; for not only does each produce its own effect, but one increases the effect of the other: namely, the obstacle adds so much to the pressure of the circulation on the heart, that this organ labours under a double disadvantage, first, from its own diminished power, and secondly, from a preternatural pressure upon it. Thus the resulting effect of the obstacle and the dilatation of the heart combined, is greater than the sum of the two taken separately.

In further invalidation of M. Bertin's opinion, I may add that I have seen numerous instances in which all the phenomena of an obstructed circulation were occasioned by dilatation alone; as no other obstacle capable of accounting for them, could be detected in the course of the circulation.

According to the foregoing arguments, then, it appears first, that dilatation may be a *primitive* disease; and that, as such, it is capable of producing all the phenomena of an obstructed circulation. Second, That when it is consecutive to another lesion, it plays a prominent, and perhaps even, in some cases, a more important part than that lesion, in producing the phenomena of an obstructed circulation.

As, in cases of dilatation combined with a mechanical obstacle, it is impossible to assign to the dilatation and the obstacle, the exact proportion which each bears in the production of the same effects, it is necessary, in order to ascertain the real effects of dilatation, to confine ourselves, in studying them, to the simple, uncomplicated form of the disease.

Taking into consideration this form alone, and admitting, on the foregoing grounds, that it is capable of producing all the phenomena of an obstructed circulation, we have next to inquire how or by what mechanism, it produces them. To answer this question,—it produces them by putting the muscular fibres of the heart preternaturally on the stretch, whereby their contractile power is diminished: they lose, as it were, in force what they gain in length; and it is this deficiency of power in the main spring of the circulation which constitutes the obstacle, if it may be so called, to the circulation; in the same way that weakness of the spring of a time-piece retards its movements.

It must be distinctly understood that these observations do not apply to dilatation with which a predominant degree of hypertrophy is conjoined, for the heart then acquires more force in virtue of the hypertrophy, than it loses by the dilatation, and the consequence is, an increased, instead of a diminished energy of the circulation. Less hypertrophy than is generally supposed, suffices to occasion this increased energy. It is not even essential that the walls of the heart be thickened at all, provided the muscular fibre is healthy, and the dilatation moderate; that is, not so excessive as to be greatly out of proportion to the thickness of the walls. It is in consequence of such cases being attended with an increased energy of the circulation, that it has been necessary to transfer them from the class of dilatation to that of hypertrophy, where they constitute the variety called *hypertrophhy by increased extent, without thickening, of the walls.*

M. Bertin conceives a case in which the heart gaining in virtue of its hypertrophy, precisely as much as it loses by reason of its dilatation, there results a sort of compensation or equilibrium, which maintains the functions in their healthy condition.* It would be erroneous, however, to suppose that this is not a state of positive disease; for, though the functions may be adequately performed while the circulation is tranquil, whenever it is hurried, the heart, either unable to contend with the increased pressure of the blood, becomes gorged; or, struggling against, and surmounting the obstacle, it palpitates violently, contracts beyond its normal degree, and expels an excess of its contents with preternatural force. In either case the lungs become congested—in the former, from retardation of the blood in the pulmonary veins—in the latter, from an excessive influx through the pulmonary artery—and in either case an attack of dyspnœa is the consequence. I have seen a considerable number of cases which I believe to have been of this description, as the patients experienced the attacks only at long intervals, and never without the application of an adequate exciting cause, returning to their ordinary state of good health soon after the paroxysms had subsided.

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* Bertin, p. 385.

SECTION IV.

SIGNS AND DIAGNOSIS OF DILATATION.

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IN the preceding section I have shown that the effect of dilatation is, to enfeeble the heart and thereby occasion the phenomena of an obstructed circulation. We have now to examine those phenomena as signs of dilatation.

The heart when dilated is subject to palpitations of a feeble, oppressed kind, and more or less distressing, frequent, and prolonged, according to the extent of the malady. In general they are more protracted than in other diseases of the organ. The attacks are provoked by any over-exertion or mental excitement.

The pulse is soft and feeble, and, if the debility of the heart be very considerable, it is small. Irregularity and intermittence are rare except when the vital powers are much exhausted, as in the extreme stage of the disease, or during a protracted and distressing paroxysm of dyspnœa. The languor of the arterial circulation causes the extremities and surface to be chilly, the disposition to be melancholy, and the character to be deficient in energy.

The blood, not being freely transmitted by the left ventricle, accumulates in the lungs by retardation; whence difficulty of respiration, cough, sooner or later attended with copious expectoration of thin, serous mucous; œdema of the cellular tissue of the lungs greatly aggravating the dyspnœa; terrific dreams with starting from sleep; and passive pulmonary hæmorrhage of dark, grumous blood in small quantities, forming sanious sputa and generally the precursor of death in individuals affected with great difficulty of respiration.

The lungs being obstructed, the engorgement is propagated backwards to the right side of the heart, to the great veins, and finally to all their ramifications. From this venous engorgement arises a series of striking phenomena which we shall review successively.

1. *Serous infiltration.*—This generally makes its appearance first in the lower extremities, because it is in them that the circulation is most languid, the return of the blood being opposed by its gravity, while it is little promoted by the action of superincumbent muscles. Increased serous exhalation takes place from the serous membranes also: whence hy-

drothorax, hydro-pericardium, and ascites, one or other of which is almost invariably present when there is much external dropsy.

2. *Discoloration of the face.*—If the complexion was originally florid, it becomes purple or deep violet, principally on the cheeks, the end of the nose, and the lips, with intumescence of the latter. If originally pale, it becomes cadaverously exsanguine, and has a dusky, leaden cast, especially about the eyes. The lips are either livid, or totally colourless. Lividity sometimes shows itself in the extremities as well as in the face.

3. *Congestion of the brain.*—This produces sub-apoplectic symptoms; as dull headache, felt principally along the course of the great sinuses; hebetude of the mental faculties; stupor, convulsions, and eventually complete coma. It is not unusual for these symptoms to supervene a few days before the fatal termination. Sometimes they depend, not on congestion alone, but partly also on serous effusion into the ventricles. This, however, is not always the case, as I have ascertained by several dissections.

4. *Injection of the mucous membranes.*—It is common to find them after death so vascular as to present the appearance of inflammation. This is especially the case in the stomach and intestines, and it is necessary to be aware of the circumstance,

in order to guard against the error of attributing the redness to inflammation.

5. *Passive hæmorrhage.*—This takes place from the lungs, as already stated: also from the nose, the stomach, the intestines, and more rarely from the bladder. It results from engorgement of the mucous membranes. The effusion consists of dark blood exuding in small quantities. When from the stomach, it has occasionally the appearance of coffee grounds.

6. *Congestion and enlargement of the liver.*—This is so common a consequence of retardation of the blood on the right side of the heart, that few persons so affected in any considerable degree, are exempt from it. By the obstruction which it occasions in the system of the vena porta, it leads to ascites.

General signs of dilatation of the right ventricle.—The signs which Corvisart regards as the most certain, are, greater dyspnœa than in affections of the left ventricle, a more marked serous diathesis, more frequent hæmoptysis, and a greater lividity of the face, sometimes reaching a dark violet hue. There is no doubt that these are effects of dilatation of the right ventricle; but they are not indicative of that affection in particular, because they are produced equally by hypertrophy with dilatation of the same cavity, and by valvular contraction on the left side of the heart. In any of these affections the colour is not an essential sign; for it depends upon the original complexion; and so far from being always livid or purple, it is very common in cases of great dilatation of the right ventricle, to see the face deadly pale and the lips exsanguine.

The sign which, with Laennec, I think the most constant and characteristic of the *equivocal* signs of dilatation of the right cavities, is, permanent turgescence of the external jugular veins *without sensible pulsation*. This turgescence does not disappear when the vein is compressed at the upper part of the neck.

Although all these signs of dilatation of the right ventricle are equivocal of themselves, they have much weight when coinciding with the evidence of auscultation; and by the two classes of signs combined, dilatation of the right ventricle may generally be detected with certainty. Whether the dilatation be connected with valvular contraction on the left side of the heart, is an ulterior question, to be determined by ascertaining whether there exist the characteristic signs of that contraction. (Vid. the chapter on disease of the valves.)

General signs of dilatation of the auricles.—This affection presents no general signs distinguishable from those of the disease in the corresponding ventricle or valve, to which it owes its origin; but its existence may safely be inferred when the valve in question is either much obstructed or permanently open, or when, from any cause, there is great retardation of blood in the ventricle.

PHYSICAL SIGNS.—The signs of the two first varieties of dilatation; namely, that with a thickened, and that with a natural thickness of the walls, are given in the chapter on hypertrophy, p. 224-5 and 229. It only remains for me to describe the signs of the third variety, or *dilatation with attenuation.*

The impulse.—In this variety the impulse is diminished, and in extreme cases, entirely absent, even during palpitation. When felt, it is only a brief percussion of the thoracic parietes not elevating the ear. When the dilatation is great, the impulse is a little lower down than natural. It sometimes happens that, of several beats of the heart that are *heard*, one only is *felt*, and if this is vigorous, it warrants a conclusion that the parietes are little attenuated. Though Laennec does not make this observation, I have assured myself of its accuracy by numerous post mortem examinations.

The sounds.—When the walls of the ventricles are merely thin without being dilated, the first sound (i. e. that produced by the systole of the ventricles) is louder, shorter, and clearer than natural; it approximates in its character to the second sound, (i. e. that produced by the diastole of the ventricles,) which is analogous to the flapping of a pair of bellows. When there is dilatation, even in a moderate degree, the first sound becomes almost the same and nearly as strong as the second; and, finally, when the dilatation is considerable, the two sounds cannot be distinguished either by their nature or intensity, but solely by their respective relations of synchronism or anachronism with the arterial pulse; and, as the pulse in remote arteries, as the radial, is often, in dilatation and various other diseases of the heart, later than the ventricular systole, the pulse of the carotid or subclavian should be felt.

In proportion as the sounds of the heart are louder, they are audible at a greater distance over the chest: accordingly, M. Laennec has proposed a scale by which the extent is made an index of the degree of dilatation and attenuation. Before describing this scale it is necessary to acquaint the reader with the range of the sounds in the natural state.

In a healthy man, of medium stoutness, and whose heart is in the best proportions, the sounds, according to Laennec, are audible in the præcordial region alone; that is, in the space comprised between the cartilages of the 4th and 7th left ribs and underneath the inferior half of the sternum;

also, if the sternum be short, in the epigastrium. I have generally thought that they may be heard a little beyond this range. The sounds are similar and equal on the two sides of the heart, those of the right being most audible under the sternum, and those of the left, under the cartilages of the ribs. When audible beyond the limits mentioned, they are heard successively in the following places, constituting the scale alluded to: viz.

1st. Along the sternum and at the left superior anterior part of the chest as high as the clavicle;

2d. Over the same extent on the right side;

3d. The left side of the chest, from the axilla to the region of the stomach;

4th. The right side over the same extent;

5th. The posterior left side of the chest;

6th. The posterior right side.

The intensity of the sound is progressively less in the succession indicated, provided the parts around the heart are in the same relative states. But there are so many diversities in these, which may interfere with the order described, that I have found the scale of M. Laennec of little practical utility in estimating the degree of dilatation. Thus, in very fat subjects in whom the impulse of the heart is not perceptible to the hand, the space over which its sounds can be heard by the cylinder, is much more limited than natural: Laennec has even found them confined, in some instances, to a square inch, though I cannot say that this has occurred to myself. On the other hand, in meagre persons, in those who are narrow-chested, and in children, the sounds are audible much further: namely, over the two inferior thirds, or even three-fourths of the sternum, sometimes even over the whole of that bone and at the left anterior superior part of the chest as high as the clavicle; often, also, though less distinctly, below the right clavicle. In very meagre subjects I have heard them over the whole chest both posteriorly and anteriorly. Now, as it is almost impossible to make an exact estimate of the degree in which stoutness limits, and leanness &c. extend, the range of the sounds, this range is not a sure criterion of the degree of dilatation.

Again, a lung in any way consolidated, whether by hepatization, tubercles, or compression by fluid in the cavity of the pleura, transmits the sounds of the heart more strongly than a lung that is sound and permeable to air—a phenomenon explicable on the principle that dense bodies are the best conductors of sound. The effect is the same though there be cavities in a tuberculous lung; for the sound is transmitted, not through the cavities, but through their walls, which are denser than healthy pulmonary substance.

Under these various circumstances, then, the sounds are irregularly propagated, and the progressive scale of Laennec is interfered with. Thus, if the right lung be consolidated, the sounds will be more audible on that side than on the left.

My own mode of estimating the degree of dilatation is, by observing how far the first sound resembles the second, and comparing the intensity of the first, heard immediately over the ventricle affected, with what I conceive from experience would be its intensity in the same subject if the heart were healthy. I then corroborate the estimate, if necessary, by the scale of Laennec; making allowance, as far as is practicable, for stoutness, leanness, youth, pulmonary condensation, &c.

The manner in which I judge of attenuation by the first sound, is less by its loudness, than by its greater shortness and clearness; for I think it is often louder in dilatation with hypertrophy or even with a natural thickness of the parietes, than with attenuation. This opinion is opposed to that of Laennec, who "thinks he may regard it as constant that the extent over which the beats of the heart are audible is in the direct ratio of the feebleness and thinness of its walls." So far is this from being perfectly true that I have met with cases in which the heart was dilated and attenuated to the extreme, yet the first sound was feeble.

Nor should we expect it to be otherwise in such cases; for when the heart, from extreme dilatation, is too feeble to contract smartly, its sounds must necessarily be weak. Hence they are so in ramollissement, and in the moments preceding dissolution.

Resonance on percussion.—The resonance of the præcordial region on percussion is diminished by dilatation. The dulness is situated rather lower down than natural, and, as it is always in proportion to the increase of volume of the heart, it is greatest in hypertrophy with dilatation.

Dulness of the præcordial region on percussion may exist independent of enlargement of the heart: namely, when the anterior borders of the lungs are hepatized, and extend in front of the heart. I have met with a case of this kind in which the hepatized borders, forced completely over the heart by emphysema of the posterior parts, not only caused defective resonance, but prevented the impulse of an enormously hypertrophous heart from being perceptible. On the contrary, dilatation sometimes does not occasion deficient resonance when the lungs are emphysematous and their anterior margins are forced between the organ and the sternum.

Physical signs of dilatation of the auricles.—Auscultation has not hitherto supplied any *direct* signs of dilatation of the auricles; but as this affection is in general the consequence of disease of the valves, and of enlargements of the ventricles impeding the circulation through the heart, its existence may be inferred from the physical signs of these affections. Thus, when there is a contracted, and, still more, a permanently open state of either auriculo-ventricular orifice, dilatation of the corresponding auricle is almost certain: and when there is hypertrophy and dilatation of the right ventricle with much jugular congestion, dilatation of the right auricle is highly probable.

SECTION V.

PROGRESS, TERMINATIONS AND PROGNOSIS OF DILATATION.

Effects of a naturally thin Heart on the Constitution, 278. *Slight Dilatation not very formidable—its Effects*, 278. *Often remains stationary*, 278. *More considerable, always tends to increase*, 279. *When Dropsy recurs pertinaciously, a fatal Termination is at hand*, 279. Prognosis, 279.

In many persons the heart, without being dilated, has naturally thin walls; that is to say, (to assume a standard of comparison for an object which cannot have any fixed one,) the walls of the left ventricle are not, at the utmost, more than twice the thickness of those of the right. This state presents signs similar to those of dilatation, but in a less degree;

namely, the impulse is diminished, the first sound is loud, short, and clear, and both sounds are more extensively audible than is natural. Individuals so affected may live for a great number of years, even to an extreme old age, in a state of tolerably good health: it is only to be remarked that this conformation is in general accompanied with a delicate constitution, a slim stature, and small muscles. In fevers and diseases of the respiratory organs, the individuals in question experience, cæteris paribus, greater dyspnœa than others. If such a conformation augments even slightly, a dilatation of the heart is the result.

A slight degree of dilatation is not a very formidable affection. The dyspnœa is sometimes not so great as to deserve the name of *morbid;* but the patient has simply a shorter respiration than most men, he more readily loses breath, and he experiences palpitations from much slighter causes. With these slight symptoms, however, he generally exhibits some traces of the cachexy proper to organic disease of the heart.

This state, which is that of a great number of *asthmatics*, may subsist very long without occasioning any disorder of a serious nature, it may remain without making progress for a great number of years, and it does not always prevent the patient from attaining an extreme old age.

When dilatation has advanced so far as to occasion *morbid* dyspnœa, it has a constant tendency to increase, unless the circulation be kept tranquil by a very quiet life and judicious medical treatment when necessary. With these precautions the disease may be kept stationary, sometimes for an indefinite period, if not exasperated by fevers or inflammatory affections, which, by hurrying the circulation, are eminently prejudicial.

When dropsy comes on, and, after having been removed by remedies, constantly shows a disposition to return, we may know that the dilatation tends to its fatal termination; and although the patient may sometimes rally from five, six, or even more attacks, he generally sinks in the course of one or two years or less. The progress of dilatation with hypertrophy is much more rapid, as already explained in the chapter on that subject.

Prognosis.—The general prognosis is founded on the above considerations, and is favourable so far as life is immediately concerned. The particular prognosis depends upon the degree of severity of the symptoms and the constitution of the patient.

SECTION VI.

TREATMENT OF DILATATION.

First Indication to remove the exciting Cause, 280. *How the Heart then recovers itself,* 280. *Remedies—Tranquillity, Diet, Air, Shower-bath,* 282. *When a bracing Air and the Shower-bath are injurious,* 282. *When Opiates are,* 282. *Bitters, Acids, Chalybeates,* 282. *Dyspepsia, Bile,* 282. *Antispasmodics,* 283. *Inflammations to be avoided,* 283. *Flannel and Wash-leather Waistcoats,* 283. *Treatment for Attacks of Dyspnœa,* 283. *Blood-letting to be sparingly and reluctantly employed,* 284.

THE treatment of dilatation with increased power of the heart, that is, with hypertrophy, is described in the chapter on hypertrophy. In this place I have only to speak of the treatment of dilatation with diminished power, that is, with attenuation, and sometimes with a natural degree of thickness of the parietes.

The first indication is, to remove, if possible, the exciting cause of the dilatation; and if this be done before the disease has proceeded to such an extent as entirely to deprive the muscular fibre of its resilience and elasticity, these faculties come into operation and restore the organ to its natural size. Accordingly, if the cause be an obstruction in the pulmonary circulation, as that produced by peripneumony, hydro-thorax, pneumo-thorax, emphysema, the use of wind-instruments, ventriloquism, tubercles, &c. the attention must be primarily directed to the removal of these affections and the prohibition of these habits. If the cause be, too violent exercises or passions, inebriety, occupations which, by placing the patient in a constrained posture, prevent the free circulation of the blood, as the professions of shoemaker or tailor, &c. the pernicious exercises, habits or professions must be abandoned and the passions calmed.

All the causes enumerated being of a temporary nature, the dilatation resulting from them, if not inveterate, can often be removed. But when the cause is permanent, as the contraction of an orifice of the heart, or a natural or acquired feebleness of the organ in proportion to its function, a cure of the dilatation is scarcely to be expected; but it may often be prevented from increasing, and the life of the patient may sometimes be prolonged even to its extreme limits. In such cases, therefore, the practitioner should steadily and perseveringly pursue a palliative and prophylactic treatment, having first discarded from his mind the impression no less erroneous in itself, than detrimental to the progress of medical science, namely, that organic diseases of the heart are necessarily fatal, and that therefore all treatment is unavailing.

The circulation should be kept as tranquil as possible by a quiet life, and a moderate unstimulating diet. The food, however, should be rather nutritious, comprising a little animal food or soup twice a day, in order to keep the muscular system in general, and that of the heart in particular, in good tone. The same object may be promoted by a clear, dry, bracing air, (as that of Brighton,) and the shower bath; from both of which I have seen the best effects result. Neither of them, however, have I found to suit those patients who have great pulmonary congestion with copious expectoration; as such require a warm humid atmosphere to favour expectoration and the cutaneous function, and they cannot bear the shower bath, on account of its determining too much from the surface to the heart and great vessels. Neither can they well bear opiates; as these remedies partly occasion diminished mucous secretion, and partly accumulation of that already secreted; both of which circumstances increase the dyspnœa.

The general health and strength may likewise be improved by the occasional exhibition of bitters, mineral acids, and chalybeates, with aromatics. The stomach in particular should be kept in good order; as its derangements—even a little flatulence or acidity—have a surprising effect in disturbing the action of the heart. The same may be said of the biliary secretion. When there is an unequal distribution of nervous power, indicated by hysterical symptoms, &c. antispasmodics, particularly the pilula galbani composita, and valerian, are very useful adjuncts to other remedies.

Febrile and inflammatory affections of every kind, but particularly inflammation of the lungs and bronchia, should be sedulously guarded against, and, when occurring, should be promptly treated. Even a slight pulmonary catarrh should be viewed as a serious affection. To prevent colds, and relieve the heart by keeping up the circulation on the surface, flannel next to the skin is almost indispensable; and, if the patient be chilly, as is frequently the case in dilatation, a jacket of wash-leather should be worn over the flannel during the winter.

Attacks of dyspnœa are best relieved by immersing all the extremities in warm water, a blanket being thrown round the patient to promote perspiration, and fresh cool air being admitted to satisfy the craving for breath.—While this is being done, he should take an antispasmodic draught composed of æther, laudanum, camphor, ammonia and assafætida, combined according to circumstances.* It may be repeated two or three times, at intervals of from half an hour to an hour, according to circumstances.

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* Vid. for particulars, Treatment of Disease of the Valves.

Blood-letting should not be resorted to in dilatation with deficient power of the heart, *during the paroxysm,* and merely for the purpose of relieving it. The abstraction of a small quantity has not the effect, and that of a large is inadmissible, as it does more injury by increasing the debility of the heart, than it does good by lightening the circulation. Consequently, an ultimate aggravation of dyspnœa ensues. More than once I have seen a large and indiscreet blood-letting fatal; as the patient could not rally from the exhaustion produced by the attack of dyspnœa to which that from the depletion had been superadded. If there be an absolute necessity for blood-letting, that is, if the dyspnœa be constant and cannot be relieved by any other means, the quantity drawn should not exceed six ounces at one time, and it should be drawn very slowly, in the recumbent position, and during the intervals or remissions of the fits. In this way the bleeding may be repeated, if necessary, every one, two or three months, provided it does not diminish, but, rather, increases the strength of the patient.

For the treatment of dropsy, cough, &c. I refer the reader to the chapter on diseases of the valves.

CHAPTER III.

PARTIAL DILATATION OR REAL ANEURISM OF THE HEART.

Description of a Case by Corvisart and two by M. Bérard, 285. *Laennec attributes the Disease to Ulceration of the interior of the Ventricles,* 286. *Corroborated by a Case which occurred to the Writer,* 286. *Pouch in the Mitral Valve,* 286.

THE heart may be affected with real aneurism. In a young negro, who died suffocated, Corvisart found the left ventricle surmounted by a tumour almost as voluminous as the ventricle itself, containing several layers of rather dense lymph, perfectly similar to those of aneurism of the limbs, and communicating with the cavity of the ventricle by a narrow, smooth and polished aperture.* M. Bérard has recorded two similar cases, except that the tumours were only as large as ducks' eggs. In one, a portion of the sac was formed by the pericardium and fibrinous layers within, the muscular substance being entirely deficient. The general aspect of one of these preparations examined by

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* Essai sur les Maladies du Cœur, p. 283.

Laennec, led him to believe that aneurisms of this kind result from ulcerations of the internal surface of the ventricles. This opinion is corroborated by a case that occurred to myself, (Brown) in which steatomatous degeneration had caused the formation of a canal from the aorta underneath one of the sigmoid valves and the internal membrane of the left ventricle, leading to an aneurism as large as a nut in the substance of the auriculo-ventricular septum. A similar case occurred subsequently in St. George's Hospital. In the latter, the second sound was accompanied with a bellows-murmur. In the former the physical signs were not noticed. The general signs were those of organic disease of the heart.

Moraud and Laennec have met with a kind of aneurismal pouch formed in the mitral valve.

CHAPTER IV.

SOFTENING OF THE HEART.

Anatomical Characters, 287. *Two Varieties—1st. With increased Redness*, 288. *2d. With diminished Redness*, 288. *Laennec supposes Softening to be an Affection sui generis resulting from a Derangement of Nutrition*, 288. *Bouillaud ascribes it to Inflammation*, 288. *The latter Opinion the more correct*, 289. *Evidence that Softening with increased Redness may be inflammatory*, 289. *Is it the Cause of the quick Pulse during Convalescence from Fevers?* 290. *Pale Softening may be either inflammatory or not*, 291. *It causes a pale sallow Complexion*, 291. *Softening leads to Dilatation*, 291. SIGNS AND DIAGNOSIS. General. *Not distinguishable from those of the co-existent inflammatory Affections*, 292; *or Chronic Disease*, 293. *A Series of Signs generally indicative of Softening*, 293. Physical. *Impulse—Sounds*, 293. *Not very Pathognononic*, 294. *How to avoid some Fallacies*, 294. PROGNOSIS. *How far the Affection is curable*, 294. TREATMENT, 295.

SOFTENING of the heart presents the following anatomical characters. The organ, when placed on a table, does not maintain its round form, but sinks and becomes flattened. When the ventricles are opened by an incision they collapse even though thickened. The muscular substance feels flaccid and tears with great facility. Sometimes it is so soft and friable, as easily to break up under slight pressure of the fingers. The softening is almost invariably accompanied with a change of colour; and as this differs in different cases, apparently according to the nature and cause of the malady, it has been made the basis of a distinction into two species; 1st. Softening with increased intensity of redness—namely, claret, morone, or violet-coloured, denoting an excess of blood in the muscular substance. 2. Softening with diminution of redness, namely, faint yellow or fawn-coloured, aptly compared by Laennec to the tint of the palest dead leaves, and bespeaking a deficiency of blood.

Softening may pervade the whole, or only a portion of the heart, and may co-exist with any other lesion of the organ.

Both M. Laennec and MM. Bertin and Bouillaud recognize the two species described, and they agree in their account of the circumstances under which they respectively occur; but they differ in their opinion as to the nature and cause of the affection, Laennec supposes it to be "an affection *sui generis*, resulting from a derangement of nutrition, by which the solid elements of the tissue diminish in proportion as the liquid or demi-liquid elements augment." M. Bouillaud (for it is he alone who, in his conjoint work with M. Bertin,) was the author of all the doctrines relative to inflammation contends that softening in all its varieties of colour, is a result of inflammation, because, as "softening of the brain, the uterus, the kidneys, the spleen, &c. is, in the present day, regarded as a certain character of inflammation," he could not adopt another opinion without doing violence to the laws of analogy.

According to my observation, both of these conflicting opinions are partly correct. That the first species, or that with increased redness, may result from acute inflammation, rests on incontestible evidence: it was found by Dr. Latham to exist in the unique case of extremely acute and rapid inflammation, in which pus was infiltrated throughout the whole muscular substance of the heart—a phenomenon that had never been known to occur when Laennec wrote, and which nullifies one of the strongest arguments by which that author supports his view: namely, "I think," says he, "we may regard as a general law in the economy that all the *soft* tissues *harden* by the effect of a *true* inflammation, that is to say, *tending to the formation of pus*,"—(this being the only definition of inflammation that he admits).* As, then it is proved that inflammation, according to his own definition of it, is capable of producing softening with increased redness, we may, without transgressing the laws of philosophic caution, presume that it may, though unattended with the formation of pus, produce the same effect; and this view is countenanced by the circumstances under which, according to the concurrent testimony of all parties, the species of softening in question is generally found to occur; namely, accompanying acute pericarditis, or inflammation of the internal membrane, and in adynamic fevers. In the latter, however, Laennec connects it with an alteration or putrescence of the liquids, having always, as he states, found it greater in proportion as the alteration was more decided.

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* De l'Auscult. tom. ii. p. 541.

Laennec enquires whether softening "could be the cause of the extraordinary frequency of the pulse which often supervenes during convalescence from fevers, and which sometimes persists for several weeks though the patient regain his strength and substance." Bouillaud resolves this question in the affirmative, but adds that the quickness of the heart's action can only be accounted for on the view that the softening is a genuine carditis. As there are no positive data by which this question can be determined, I leave the reader to judge for himself.

The second species of softening, or that with a pale or yellowish colour, has generally been found in subjects who have long been in a cachectic state, or who have been worn down by slow or hectic fever, or, finally, who, at no very remote period, have had pericarditis. I have no doubt that the pale colour may in some cases result from inflammation, as I have seen it penetrate only a certain depth into the muscular substance, as if propagated from the inflamed pericardium which had become adherent. But I am disposed to believe with Laennec, that in cases of cachexy and marcor, it may occur independent of inflammation. For I have repeatedly met with instances of enlarged heart, in which the organ was universally pale and flaccid, yet no inflammatory or febrile affection had antecedently existed to account for the state. The affection therefore appears to have been referable to the same causes as, in such cases, sometimes render the other muscles pale, flaccid and withered. Laennec remarks, and I suspect with great truth, that, when there exists cachectic softening, the complexion is always pale and sallow; and that even when it accompanies dilatation or hypertrophy, the lips are rarely violet or bloated, but almost always nearly colourless.

As softening diminishes the cohesion, and therefore the elasticity of the heart, we are necessarily led to infer that it conduces to dilatation: accordingly we find that dilatation is its almost constant concomitant, when it has subsisted for a considerable period.

Signs and diagnosis of softening.

General signs.—As softening from acute inflammation generally co-exists with pericarditis or with adynamic fever, there is difficulty in distinguishing its signs from those of the other maladies. Complicated with them, it is attended by a quick, feeble, small, and faltering pulse, great anxiety, and a disposition to syncope—the same symptoms, in short, that characterize pericarditis with copious fluid effusion. Now, as such an effusion is generally present when the inflammation is so severe as to affect both the pericardium and the muscular substance, it is scarcely possible, in every case, to say positively whether the severe symptoms in question depend on the effusion or on the softening. I am disposed, however, to think that the latter, as well as the former, is capable of producing them; as they sometimes exist when the quantity of fluid is scarcely sufficient to constitute an adequate cause, and as it is consistent with analogy to suppose that the muscular tissue of the heart, when softened by inflammation, would, like other muscles, be rendered incapable of adequately discharging its function. In this point of view, softening greatly aggravates the severity and danger of pericarditis, and, I should imagine, of fever also.

The signs of softening from chronic inflammation or other wasting disease, as scurvy, hectic, &c., are no less ambiguous; as they may result from the disease itself, independent of softening. They are, general languor, a sallow, exsanguine, withered complexion, a quick but soft and feeble beat of the heart and pulse, gradual reduction of the strength, and dropsical effusion from sluggishness of the circulation.

I have frequently found softening after a series of symptoms mentioned by Laennec: namely, when, in a case of dilatation with or without hypertrophy, there have been long and frequent attacks of suffocative dyspnœa; when the struggle between life and death has been long,—of several weeks' duration, for instance; and when the violet hue of the face, the extremities and the other parts of the surface of the body, had announced, long before death, the retardation of the blood in the capillary system.

Physical signs.—As the systole and diastole of the heart are enfeebled by softening, its impulse is more or less reduced in strength, and both its sounds become dull and obtuse. When the affection is accompanied with acute inflammation of the organ, the contractions of the ventricles, though feeble, may still be somewhat smart or jerking. These signs are, unfortunately, not very pathognomic, for the same occur in pericarditis with copious fluid effusion.

There is also a difficulty in ascertaining how far, in cases of enlargement of the heart, the dulness of the sounds may be occasioned by hypertrophy: and, again, how far any diminution of them from softening may be neutralized by an increase which they sustain from dilatation. The following rule has appeared to me to be the best criterion: viz. when the impulse is weaker and the sounds duller than might be anticipated from the degree of enlargement, as estimated by the extent over which the heart's movements and dulness on percussion are perceptible, the organ is softened. Hydro-pericardium extends the range of pulsation and non-resonance; but as the fluid may be detected by the undulatory impulse, &c. (vid. Hydro-pericardium), a fallacy can scarcely arise from this circumstance.

Prognosis.—The prognosis of softening depends upon the co-existent and as it were primitive affection. As above stated, it greatly augments the danger of pericarditis and probably of fever, but when these affections terminate favourably, there is every reason to believe that the muscular substance may be restored to its healthy condition. With chronic maladies and organic disease of the heart, the effects of softening are less directly pernicious, but, as the exciting causes of the affection are more permanent, restoration to the healthy state is more rare.

Treatment.—When accompanied by acute inflammation, softening must be treated on the same principles as pericarditis; when a result of chronic disease, it demands the same remedies as the primary affection, and especially iron, bark, a nutritious diet, and good air, if they be not otherwise contra-indicated.

CHAPTER V.

INDURATION OF THE HEART.

Induration described by Corvisart, 296. *It is a Perversion of Nutrition,* 297. *Chronic Inflammation probably its Cause,* 297. *It increases the Impulse of the Heart,* 297. *Except when extreme,* 297. *Treatment same as Hypertrophy,* 297.

The muscular substance of the heart sometimes undergoes induration. Corvisart has found it carried to such an extent that the heart, when struck, sounded like a dice-box or hollow horn vessel, and the scalpel, on making an incision, experienced great resistance, and produced a singular crepitating noise. Yet the fleshy substance possessed its proper colour, and did not appear converted into either an osseous, a cartilaginous, or any similar substance. This affection is very rare. Laennec and Bertin have met with it affording a resistance to the scalpel, but not causing the crepitating noise; and the same has occurred to myself. It generally occupies the whole of a ventricle, but sometimes only a portion, and it may accompany any state of the organ as to size, though most commonly it is conjoined with hypertrophy.

It consists, I apprehend, not merely, as Laennec supposed, in an increase, but in a perversion of nutrition, being somewhat different from that firmness which the heart frequently acquires by hypertrophy. MM. Bertin and Bouillaud, with I think the majority of authors, regard it as one of the products of chronic inflammation.

Induration, according to Laennec, increases the impulse of the heart. The firmest hearts with which he had met, were also those which gave the strongest impulse. But it is conceivable that when the induration proceeds beyond a certain point, it must, as Corvisart thought, render the contraction of the ventricles more difficult, and their movements more limited.

The treatment of induration with increased action of the heart is identical with that of hypertrophy.

CHAPTER VI.

ADIPOSE AND GREASY DEGENERATIONS OF THE HEART.

EXCESS OF FAT. *Exists both externally and between the Fibres,* 298. *Old Authors thought it caused formidable Symptoms,* 298. *This idea in general incorrect,* 299. *Seldom causes Rupture of the Heart,* 299. GREASY DEGENERATION, 299. *Distinguished from Softening by its greasing Paper,* 300. *Atrophy and Œdema of the Adipose Tissue,* 300.

Excess of fat.—In individuals remarkable for obesity, and occasionally in others of only moderate *embonpoint,* the heart is sometimes greatly overloaded with fat, which, deposited between the pericardium and the muscular substance, not only covers the organ externally, but frequently penetrates a considerable depth between its fibres; while the walls themselves, as if losing (probably by the pressure) what the adipose tissue gained, become attenuated and flabby.

The older authors imagined that this affection was the cause of more or less severe symptoms and even of sudden death. Corvisart thinks that an enormous accumulation might sometimes produce such an effect, though, in the persons in whom he had met with very fat hearts, he had seen nothing which could prove to him " that the state was morbid, that is to say, carried to such a point as constantly to derange the function of the organ and thus constitute a malady." The experience of Laennec has led him to the same conclusions, nor have I seen any thing that invalidates them.

It would be natural to suppose, that the substitution of adipose for muscular tissue, and the extreme attenuation which the walls, especially the apex and the posterior part of the right ventricle, sometimes undergo from this cause, would be eminently favourable to rupture of the organ; yet this accident is very rarely the result. Morgagni has seen it, but Bertin has only met with a case of rupture of the auricle, while Corvisart and Laennec have not met with an instance at all. The alteration described is different from that denominated—

Greasy degeneration of the heart.—This, according to Laennec, is " an infiltration of the muscular substance with a matter which presents all the physical and chemical properties of grease; it is an alteration exactly similar to the greasy degeneration which Haller and Vicq-d'Azyr have observed in the muscles. Laennec has never found it but in a very small portion of the heart, and only near the point. It was of a pale yellowish colour, like dead leaves, and therefore very similar to certain varieties of softening; but he thinks that it may be distinguished from this, by its strongly greasing paper between which it is pressed. I have seen a remarkable case in which a degeneration of this kind, occupied the greater part of both ventricles.

Atrophy and œdema of the adipose tissue. The former sometimes accompanies general emaciation, and the latter presents itself in cases of universal dropsy.

CHAPTER VII.

OSSEOUS, CARTILAGINOUS AND OTHER ACCIDENTAL PRODUCTIONS OF THE MUSCULAR SUBSTANCE OF THE HEART AND OF THE PERICARDIUM.

Rare. Descriptions, 301. *Laennec thinks that they would increase the Sounds of the Heart,* 302. *This Opinion conjectural,* 302. *Osseous and Cartilaginous Depositions in the Pericardium,* 302. *Incurable,* 303. *Treatment palliative,* 303. *Tubercles and Cancer,* 303. *Cancer exists in two forms, 1st. Isolated Tumours, 2nd. Interstitial Infiltration,* 303. *They impede the Action of the Heart,* 304. *Serous Cysts and vesicular Worms,* 304.

Osseous and cartilaginous productions of the muscular substance are very rare. Corvisart has seen the point of the heart, in its whole thickness, and the left columnæ carneæ converted into cartilage. Burns has seen the ventricles perfectly ossified so as to resemble the bones of the cranium. Haller, Filling and Bertin have seen partial ossifications. M. Renauldin has found the left ventricle converted into a real petrifaction, which had a sandy appearance in some parts, and in others resembled a saline crystallization. Cartilaginous incrustations occasionally exist between the lining membrane and the muscular substance. Kreysig found one in an ossified state.

Laennec feels persuaded that an osseous or cartilaginous induration of a large portion of the heart, as a whole ventricle or half the organ, could be recognised with the cylinder, by a very marked augmentation, and some particular modifications, of the sound of the organ. He thinks that cases of this nature are amongst those in which the sound of the heart can be heard at a certain distance from the patient. There is little doubt that such cases would occasion a preternatural murmur: but whether this murmur could be distinguished from that of ossified valves, &c. and whether it would be so loud as to be audible at a distance from the patient, is problematical, as, in the cases that have hitherto presented themselves, these phenomena have not been explored.

Osseous and cartilaginous depositions sometimes take place in the pericardium; and though they do not properly fall amongst the diseases of the muscular substance, they are introduced here because they are not of sufficient importance to form a separate chapter. Laennec met with an osseous deposition between the fibrous and serous layers, which formed a band from one to two fingers broad completely encircling the heart, and sending off triangular processes towards the apex.* In other cases, in which the concretion has formed a similar ring, or a case nearly enclosing the whole organ, it has sometimes given off processes which penetrated the muscular substance † and reached even into the cavities. The general symptoms have been identical with those of disease of the valves creating great obstruction of the circulation.

As osseous or cartilaginous degeneration of the heart and pericardium is incurable, the treatment can only be palliatives.

Tubercles and tumours of a carcinomatous nature have been found in the substance of the heart. Recamier has seen the organ converted in part into scirrhous matter like the skin of bacon, in a subject who had also carcinomatous tumours in the lungs. M M. Laennec, Andral and Bayle, and others have found encephaloid cancer of the heart.

From these cases it appears that, in the heart, as in other organs, carcinomatous productions may be developed in two principal forms, that of *isolated tumours*, and that of *interstitial infiltration*. They rarely exist without similar productions in other organs, especially the lungs. There can be no doubt that cancer, if sufficiently extensive, would impede the action of the heart and obstruct the circulation; but the cases on record are too few to afford data for a general history of the disease. Serous cysts and vesicular worms (apparently the *cysticercus finnus* of Rudolphi) have also been found in the heart.

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* De l'Auscult. tom. ii. p. 675.

† Latham, Lond. Med. Gaz. vol. iii. p. 7.

CHAPTER VIII.

ATROPHY OF THE HEART.

Definition, Instances, and Causes, 305. *Atrophy with Contraction*, 306. *With Dilatation*, 306. *Its Effects slight*, 306. *Obviates inflammatory and predisposes to hypochondriacal Affections*, 306.

ATROPHY consists in deficient nutrition, and the heart, like any other muscle, is liable to it. The heart of an adult was found by Burns not larger than that of a new-born infant, and the heart of a female of twenty-six not larger than that of a child of six. Bertin gives a similar case: (66.) the writer has met with the same; and numerous other instances are on record.

Atrophy generally takes place under the influence of those causes which produce general emaciation: chronic diseases for instance, as phthisis, diabetes, chronic dysentery, cancer and malignant affections in general. Excessive bleeding is another cause. Laennec adduces an instance resulting from the treatment of Albertini and Valsalva employed to cure hypertrophy. Finally, protracted compression by fluid effused within the pericardium, as in cases of chronic pericarditis, may produce the effect.

The heart, when atrophous, generally contracts upon itself, so as to diminish its cavities, while its walls do not become materially thinner, and sometimes become even thicker than natural. In the latter case, the affection must not be mistaken for hypertrophy, and the error may be avoided not only by remarking the general diminution of the volume of the heart, but also the shrivelled and wrinkled appearance of its exterior.

Atrophy may also co-exist with dilatation when the walls are so thin that the total volume of the muscular substance is diminished.

Diminution of the volume of the heart does not appear to produce symptoms which entitle it to be ranked as a disease. Individuals who present this peculiarity are perhaps less subject to inflammatory complaints than others, though they are more prone to fainting from slight causes, and to hypochondriacal affections. It is remarkable that women, who are more subject to these ailments than men, have in general smaller hearts.

CHAPTER IX.

DISEASES OF THE VALVES AND ORIFICES OF THE HEART.

SECTION I.

ANATOMICAL CHARACTERS, WITH PREDISPOSING AND EXCITING CAUSES, OF DISEASE OF THE VALVES.

Natural Structure of the Valves, 307. *Fibro-serous—hence predisposed to Disease*, 308. *Lining Membrane of the muscular Substance comparatively exempt*, 308. *Depositions seem to originate in the fibrous Tissue*, 309. *State of the internal Membrane over them*, 309. *Valvular Disease more rare on the right, than the left Side—in what Proportion*, 309. *Cause of this*, 310; *according to Corvisart*, 310; *to Bertin*, 310; *to the Writer*, 311. INDURATION OF THE MITRAL VALVE, 312. *Characters of cartilaginous Disease and Effects on the Valve*, 312. *Description of Ossification mixed with Cartilage*, 313. *Of more purely calcareous Disease*, 313. *Ossification not natural to the old*, 314. *Changes of the Valve from Ossification*, 315. INDURATION OF THE AORTIC VALVES, 316. *Cartilaginous*, 316. *Osseous*, 316. *Of the* RIGHT VALVES, 318. EXCITING CAUSES, 319.

THE valves and chordæ tendineæ consist, according to the best authorities, of fibrous tissue interposed between a production and reduplication of the lining membrane of the heart. The fibrous tissue is prolonged from a dense, whitish zone of the same which encircles each of the orifices of the heart, and is, as it were, the tendon or point of attachment into which the muscular fibres of the organ are inserted. The lining membrane of the heart, according to Bichât, approximates closely in character to serous membranes: the valves, therefore, may be said to consist of *fibro-serous* tissue. This tissue in general, is remarkable for its proneness to cartilaginous and osseous degeneration; whence we derive an explanation of the fact, that the valves and orifices of the heart are frequently affected with these degenerations, while the cavities, where they are invested solely with the lining membrane, are in a great measure exempt. Though disease occupy a valve universally, it stops abruptly where the serous membrane is continued from the circular zone, or the extremities of the chordæ tendineæ, upon the muscular substance. In a few rare instances it advances farther; but I have never seen it attack the membrane of the muscular substance without its being connected with, and apparently propagated from, disease of the valves.

It would appear that the disease is more dependent for its origin on the fibrous, than on the serous tissue; for, where the former is most abundant, namely, at the base and the free margin of the valves, cartilaginous and osseous depositions are the most frequent and extensive. The depositions appear, indeed, to originate in the fibrous tissue exclusively; for it is common to find the valves encumbered with large masses of cartilage from which the internal membrane can be peeled off in its natural thin and transparent state. In these cases the surface of the morbid deposition is smooth and equable; and it is seldom until it becomes corrugated, rugged and knotty, that the internal membrane is implicated in the disease. Calcareous depositions, in the same way, seem always to commence underneath the membrane. In a case under my observation, in which two rings of bone as thick as writing quills encircled the left orifices of the heart respectively, the membrane was stretched like a blue film over the whole of the aortic, and the greater part of the mitral ring.

Valvular disease is much more rare on the right, than on the left side of the heart. Bichât, indeed, denied its existence at all in the former situation, but his opinion has been fully disproved. Morgagni, Vienssens, Hunauld, Horn, Cruwel, Corvisart, Burns, Bertin, Louis, Laennec, have all met with instances of disease of the right valves. Dr. Latham thinks that in one third of the cases in which he has seen disease of the left valves, it has existed in the right also. I have notes of eight cases in which it existed in the right, and can recollect several others. In six of the eight the left side was simultaneously affected, and generally to a much greater extent; but the proportion which the whole number mentioned, bears to the cases that I have seen of disease on the left side, is less than that indicated by Dr. Latham, not exceeding, I think, one in four and a half to five. It is remarkable that in all my own cases, and nearly all those of the authors quoted (with the exception of Dr. Latham who is silent on this point) the induration on the right side was merely cartilaginous. When the two sides are affected at once, it very rarely happens that the disease on the right, is greater than that on the left: in general, it is much less, being comparatively slight or incipient.

The cause of the remarkable difference which the two sides of the heart exhibit in their liability to induration, has not been positively determined. Corvisart attributed it to a more decided fibrous organization of the left valves, in virtue of which they are more disposed to receive the matter that is to transform them into cartilage, or the calcareous salts that impart to them an osseous or stony hardness. Bertin has ascribed the difference to the different nature of the blood that traverses the two sides respectively, the left receiving blood of a more vital, more stimulating, more irritating quality than that by which the right cavities are moistened. Laennec does not offer a decisive opinion.

I do not pretend to decide which of these two opinions is correct, and whether both causes may not conspire to produce the effect, or whether both may not be groundless. But, in any case, I believe that disease of the left valves is promoted by the greater energy of action of the left ventricle, by which those valves are more strained. This opinion is countenanced by the facts, that ossification of the arteries, particularly those of the brain, is a remarkably frequent concomitant of hypertrophy of the left ventricle; and that this affection of the ventricle is generally attended with, or productive of, a thickened state of some of the valves. (See Diseases of Arteries, p. 166.)

The characters of valvular induration are somewhat different according as the disease occupies the auriculo-ventricular, or the arterial valves; the cause of which is to be found in the difference which naturally subsists between the valves themselves. I shall therefore describe the degenerations of the two classes of valves separately. It may be premised that there is no essential difference but in degree and frequency of occurrence, between the degenerations on the two sides of the heart; consequently, a description drawn from the left will apply to the right.

Induration of the mitral valve.—The appearance presented by the indurated mitral valve differs according as the disease occupies the base, the margin, or the whole of the valve.

When the whole is affected with cartilaginous degeneration, the valve is generally contracted throughout, and what is lost in space appears, as it were, expended in thickening the free border; for this is converted either into a ring, an oval-shaped collar, or a transverse slit like a button-hole. The size of the aperture is various. I have seen it of all sizes from an inch to a quarter of an inch in its longest diameter. The thickness of the border likewise varies. I have seen it equal a writing quill. (M'Lean.) When the valve is thus contracted it generally projects more or less, in a funnel shape, into the cavity of the ventricle. In one case I found it project so far that the columnæ carneæ were inserted immediately into the ring, the chordæ tendineæ having disappeared. The surface of the induration is smooth, polished and translucent until the disease throws out osseous or other excrescences, which, interfering with the integrity of the investing membrane, render it corrugated, rugged, and opake. Before ossification takes place, the induration described sometimes presents a truly cartilaginous hardness, and sometimes the consistence of fibro-cartilage, or only that of tendon. When divided, the aspect of the section varies according as the disease is cartilaginous, fibro-cartilaginous, or tendinous.

In a more advanced degree, cartilaginous induration is transformed into imperfect bone. It seldom happens, however, that more than a very small proportion of the cartilaginous mass is ossified, and the change takes place sometimes at its surface, and sometimes deep in its substance. The bone produced does not exhibit the fibrous structure and peculiar arrangement of natural bone; though, as it contains a large proportion of cartilage, it may be presumed to possess more or less vascularity and vitality.

There is another species of osseous induration of the valves, which is essentially different from the above, inasmuch as it consists of calcareous matter in great predominance, and, like vesical calculi, has no vitality. It presents itself under the form of small, polished, and semi-transparent scales; or of minute, yellowish, opake granules, the agglomeration of which forms concretions of various dimensions, from a mere point to the size of a horse-bean. The deposition commences underneath the lining membrane, and generally in a small patch of indurated, cheese-like matter, the surrounding parts being healthy. The scales lie flat and superficial under the membrane, while the granules penetrate more or less deeply into the subjacent tissues. When either the scales or the granules enlarge, and their surfaces become rugged or acuminated, they cause absorption of the internal membrane, and come in immediate contact with the blood.

Some authors believe that ossifications of this description are *natural* to old people, because they occur in the majority of those who have attained the age of sixty. Whatever be the character of the ossification, whether it be mixed with cartilage or purely calcareous, to me it appears to be a morbid production. The circumstance of its occurring in the majority of persons above the age of sixty, does not militate against this view: for, as the elasticity of the arterial, as of all the other tissues, is diminished by age, the valves of the heart and the coats of the arteries are, in the aged, less capable of resisting the distending force of the blood, and are therefore more liable to disease. Nor does the circumstance of the ossification being more calcareous and less cartilaginous in the old than in the young, prove that, in the former, it is a *natural* change. It confirms, indeed, what is proved by every part of the bony tissue; viz. that in age the ossific tendency is greater; but it does not, for this reason, follow that the tendency is natural when it displays itself in an unnatural situation, as in the heart and arteries.

Sometimes the membranous portion and free margin of the valve are healthy, while the fibrous zone at the base is cartilaginous, or beset with small calcareous incrustations, or, as sometimes happens, its whole substance is converted into a thick ring of bone. By these depositions at the base of the valve, the orifice is more or less contracted, while the valve itself may remain capable of closing. In many cases, again, the base and middle are sound, and the free margin alone is diseased, its conical processes forming adhesions with each other and contracting the circumference of the valve to such an extent as almost completely to close the orifice. (Mrs. —l—n.) It is not uncommon to find the margin studded with small cartilaginous nodules, or roundish calcareous granules, which prevent the accurate adaptation of the edges to each other, and allow regurgitation during the ventricular contraction. Sometimes, the only diseased appearance that the valve presents, consists in brittle scales or patches of pure phosphate of lime between the two component layers of the membranous portion, which they occasionally rupture, and thus come in immediate contact with the blood.

Induration of the aortic valves.—Induration of the aortic valves, like that of the mitral, is more frequent and extensive at the base and free border, than in the intermediate space. At the border, it originates more especially in the corpora sesamoidea, and these bodies are sometimes enlarged by cartilage to the size of peas. I have seen the margin contracted by fibro-cartilage into a ring a quarter of an inch in diameter. (Hedgley.) The valves are sometimes thickened, nodulated and corrugated by an opake yellow degeneration, consisting of a mixture of cartilaginous and steatomatous matter. I have seen the angles of the valves detached from their bases and partially wasted away by this degeneration; so that, adhering by their centres only, they hung loose into the artery and were destitute of fulcra by which to oppose the reflux of blood from the aorta. (Copas.) In another instance, the same disease had undermined and more or less detached the bases of all the valves throughout nearly their whole length; and, under one of them, it had led to the formation of a canal, as wide as the little finger, beneath the lining membrane of the heart, leading to an aneurism in the muscular substance of the septum between the left auricle and ventricle (Case of Brown).

Such are the cartilaginous and steatomatous degenerations of the aortic valves. The osseous, of which we have next to speak, are perhaps even more frequent in the aortic, than in the mitral valves. The ossification may be either pure, or combined with cartilage. In one case under my observation, an irregular, scabrous, and denuded concretion, the size of a pea, occupied the edge of one of the valves and projected into the cavity of the artery. (Porter.) In another case, a similar mass, of a conical shape, sprung from the base between two of the valves, and presented its apex towards the centre of the vessel. (May.) Smaller concretions of this description and in this position, are common. M. Bertin saw an ossification of one of the aortic valves which had attained the size of a pigeon's egg.* In one of my cases, already alluded to, the fibrous zone encircling the base of the aortic orifice was converted into a ring of bone as thick as a quill.

When the ossification is confined to the margin and base, while the middle portion is still healthy over a certain extent, the valve, if its thickening is not very considerable, may still rise and fall, and not offer any marked obstacle to the circulation. But when the ossification pervades the middle portion of the valves, they shrink, become soldered together, or curl up upon themselves, in the direction either of their concavity or convexity, so as to present a rude representation of certain sea-shells. In this state, they may become immoveable. If curled forwards, they remain applied along the walls of the aorta, and oppose no other impediment to the course of the blood than what results from the thickness of the ossification. If curled backwards, they remain fixed in the fallen or shut position, and considerably contract the orifice. Not unfrequently, one of the three valves is curled in an opposite direction to the other two. Corvisart has seen all three ossified in the closed position, and they would only have left an extremely narrow cleft for the passage of the blood, had not one retained sufficient mobility at its base to perform a movement which augmented, by a line or two, the width of the cleft.

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* Bertin Obs. liii.

Induration of the valves on the right side of the heart.—Induration of the right or venous valves is, as already stated, almost always simply cartilaginous or fibro-cartilaginous, and is comparatively rare, not existing in perhaps more than about one case to five of disease in the left valves. It seldom presents itself without being accompanied by disease of the left valves also, and it is, in general, less advanced than the latter. (Anderson. Sharpe.) The tricuspid is more frequently affected, than the pulmonic valves. I have never seen the latter diseased, but I have once found them incapable of closing the orifice in consequence of dilatation of the artery, (Weatherly) and I have seen the orifice contracted to the diameter of a quill, an inch below the valves (Collins). M. Bertin has seen the valves themselves contracted into a circular aperture only two lines and a half in diameter. As already stated, disease of the right valves whether cartilaginous or osseous, only differs from that of the left in frequency and extent, its characters being essentially the same.

Exciting causes of valvular induration.—These are, first, such as overstrain the valves by increasing the force of the circulation; namely, violent efforts, hypertrophy, increased action of the heart from nervous, febrile, or inflammatory excitement: secondly, inflammation of the internal membrane of the heart, resulting from carditis, pericarditis—especially rheumatic, from fever or from any other cause. It would be an unnecessary repetition to dwell on this subject, as I have treated of it in the chapter on Arteritis, vid. p. 162.

SECTION II.

ANATOMICAL CHARACTERS, AND CAUSES OF WARTY VEGETATIONS OF THE VALVES.

Form, Size, Surface, Collocation, Number, 320; *Colour, Texture, Consistence,* 320. *State of the Internal Membrane,* 321. *Vegetations from an unbroken Surface,* 321.

From a Broken, 321. *Spring especially from the Base and free Margin of the Valves*, 321. *Most frequent on the left Side of the Heart*, 321, *and particularly on the Aortic Valves*, 321. *Occur occasionally in the Auricles*, 321. *Laennec thinks Vegetations are polypous Concretions*, 322. *Objections to this View*, 322. *Kreysig, Bertin and Bouillaud ascribe them to Inflammation*, 323. *This View perhaps partly correct*, 323. *Corvisart thought them Syphilitic*, 323. *This Opinion untenable*, 323.

These excrescences bear a close resemblance to venereal warty vegetations on the external organs of generation. Their form is in general irregularly spherical, oval, or cylindrical: their size varies between that of a small pin's head and a large pea, but when isolated they are occasionally as large as a horse-bean. Their surface is polished, but often lobulated like a raspberry: they are found either isolated, in clusters, or in closely agglomerated patches like cauliflowers. Their number is various: sometimes there are only one or two, and sometimes they pervade the whole of the valves, the tendinous cords and a great portion of the auricle. (Dolan.) Their colour, occasionally of a greyish or yellowish white, is more commonly heightened, universally or in parts, with pink or red of greater or less depth. Their texture is fleshy and slightly translucent, like the exuberant granulations of an ulcer. Their consistence is variable; in general they are soft and humid, as if only recently and imperfectly organized; and they can then be easily scraped off with the handle of the scalpel; but sometimes they are firm, and cannot be detached without tearing with the nail or cutting with the edge of the scalpel. Firm vegetations are generally larger and more truly warty than soft.

The internal membrane of the part from which vegetations spring, is almost invariably more or less diseased. It is thickened, steatomatous or cartilaginous, ossified, ulcerated or ruptured. When vegetations grow from a diseased, but *unbroken* surface, they may be numerous and occur in several parts at once; but when they grow from a ruptured or ulcerated edge, they are few in number, often not exceeding one or two, are generally confined to that edge exclusively, and attain a larger size than any others. I have seen them exceed a horse-bean and with a neck two, three or four lines long. It cannot be doubted that their origin is connected with the broken state of the membrane.

The base and free margin of the valves appear to be peculiarly favourable to the growth of warty vegetations. Along these parts, but especially the latter, they are often arranged in a single row. They occur on both sides of the heart, but less frequently on the right. The aortic valves are the parts most subject to them. They are more rare in the auricles than on the valves, especially in the right auricle. I have seen one third of the left auricle completely covered with them. (Dolan) When situated at the base, or the free margin of a valve, they contract its aperture according to their size and number.

Laennec thought it "indubitable that vegetations were nothing more than small polypous or fibrinous concretions, which, being formed on the sides of the valves or auricles, become organized by a process of absorption or nutrition analogous to that which converts albuminous false membranes into adventitious membranes or cellular tissue." This opinion is unsatisfactory, for as polypi are most common in the right cavities of the heart, vegetations ought to be so likewise,—the reverse of which is the fact. The valves, moreover, being perpetually in motion, would be the last parts to which albuminous concretions would adhere, as it is a stagnant state of the blood which is most favourable to their formation; yet the valves are the parts most subject to them. We most commonly find *real* albuminous concretions of small size amidst the intricacies of the columnæ carneæ, where the blood is more stagnant than elsewhere. Finally, if vegetations were merely fibrinous concretions, instead of being rare, they ought to be frequent; for, as the circumstances which, on this view, lead to their formation, are common to all persons labouring under an obstructed circulation, all, or to say the least, many, should be affected with them.

Kreysig attributes their formation to inflammation. MM. Bertin and Bouillaud have espoused the same opinion, resting on the fact that vegetations bear a close analogy to the albuminous granulations occasionally found on serous membranes affected with chronic inflammation. The small and soft vegetations certainly do bear this analogy—a fact of which I have satisfied myself by comparing the two as occurring in the same subject; but the like cannot, in the least degree, be said of the large and more properly wart-like vegetations; whence it is to be inferred that inflammation *alone* is not their cause. I am disposed to think that it is inflammation modified by some other morbid action dependent either on the constitution, or on previous structural lesions of the parts affected.

The resemblance which the firmer valvular vegetations bear to venereal warts, led Corvisart to think that they might have the same venereal origin. This opinion, however, is not tenable, as extensive observation in venereal hospitals has proved that vegetations of the heart are not more common in persons affected with this disease than in others; and it is certain that they have occurred in some who had never been in the least degree tainted with the disease.

SECTION III.

PATHOLOGICAL EFFECTS OF DISEASE OF THE VALVES, AND MODE OF THEIR PRODUCTION.

Valvular Diseases obstruct the Circulation and thus produce their Symptoms, 324. *When severe, they result partly from Disease of the Muscular Substance*, 324. *This View substantiated*, 325. *Practical Importance of this View*, 326. *How Dilatation aggravates Symptoms of Valvular Disease*, 326. *How Hypertrophy*, 327. *Danger of a wrong View of this Subject*, 327.

Diseases of the valves, whatever be their nature, whether osseous, cartilaginous, or warty, have for their common effect, to obstruct the orifices of the heart; and this they do, either by contracting the apertures, or by encumbering the valves in such a manner as to prevent them from opening and closing with suitable accuracy and facility. A mechanical obstacle is thus presented to the circulation, and from the obstruction and embarrassment which it occasions, are derived the symptoms of valvular disease.

The general symptoms, however, when of an aggravated nature are seldom dependent on the valvular obstruction exclusively; they are partly attributable to a co-existent disease of the muscular apparatus of the heart. For, so long as the organ remains free from dilatation, hypertrophy, or softening, the valvular disease, according to my observation, is not in general productive of great inconvenience.

This opinion is founded on the following grounds. I have seen individuals, who were affected in an eminent degree with disease of the valves or of the aorta, maintain for years a very tolerable state of health so long as there was no hypertrophy or dilatation of the heart: but, in proportion as these supervened, the symptoms of valvular obstruction became more and more developed, and eventually assumed their most aggravated form.

I have reason to believe that, in these cases, the symptoms were attributable in a great measure to the hypertrophy or dilatation, because I have seen a greater valvular contraction produce less severe symptoms when the hypertrophy or dilatation was less considerable. It might be supposed that a great degree of contraction would *of itself* suffice to produce the symptoms of an obstructed circulation in their most aggravated form. This is highly probable, but it does not admit of demonstrative proof, as a great degree of contraction is perhaps never found on dissection without hypertrophy or dilatation. I therefore infer that these affections ensue as consequences of valvular contraction, and I believe, for the reasons above assigned, that they play an important part in the production of the symptoms.

It is of immense practical importance to keep in view the facts stated, namely, that valvular contraction does not produce formidable symptoms until it has given rise to hypertrophy or dilatation; and that it invariably leads to these affections unless the circulation be kept tranquil. We thus know that the most efficacious treatment of valvular disease consists in employing such prophylactic measures as are calculated to prevent the supervention of hypertrophy or dilatation, and employing them with the same uncompromising strictness before those affections have appeared, as if they actually existed.

It remains to be explained how dilatation and hypertrophy aggravate the symptoms of valvular obstruction. I have elsewhere* shown, that dilatation of the heart, by enfeebling the contractile power of the organ, constitutes as truly an impediment to the circulation, as a more direct mechanical obstacle. When, therefore, dilatation exists in addition to such mechanical obstacle, it is clear, that the symptoms, having a twofold cause, must be doubly severe.

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* Vid. Dilatation, p. 263.

Hypertrophy aggravates the symptoms of valvular obstruction, because the heart, being morbidly irritable, struggles against the obstacle and falls into fits of palpitation; and as, during these, a greater quantity of blood than natural has to be transmitted through the contracted aperture, the circulation is performed with increased difficulty.

It is in consequence of these reciprocal reactions of the valvular, and the muscular apparatus on each other, that cases thus complicated are more severe than any others.

From what has been said here and in the chapter on dilatation, the reader will judge how totally some authors have been wrong in referring the obstruction of the circulation to the valvular contraction exclusively, without allowing that the enlargement of the heart contributes in any degree to the effect. Such a doctrine is not only erroneous, but dangerous, as it leads to pernicious practice. For, imagining the valvular contraction to be the only formidable part of the complaint, to it alone those authors direct their attention; and, acting on the inaccurate presumption that it is in *all* cases caused by, and accompanied with inflammation, they attack it with blood-letting, general and local, abstinence, digitalis, &c.—means which cannot remove valvular contraction when once formed, (as must always be the case before the symptoms can exist,) and which are, therefore, a useless expenditure of the patient's strength. It is true, indeed, that measures calculated to diminish the force of the circulation are useful in obviating the supervention of hypertrophy or dilatation—the paramount source of danger in these cases;—but measures employed for this purpose and which must be continued for an indefinite length of time, cannot be practised with the same activity as for the purpose of curing an inflammation. I would not be understood by this to mean, that valvular disease is *never* accompanied by inflammation, and that, when so accompanied, it should not be treated by antiphlogistic measures: but I mean that they should not be employed unless there is reasonable evidence of inflammation.

SECTION IV.

SIGNS AND DIAGNOSIS OF DISEASE OF THE VALVES.

GENERAL SIGNS. *Those common to Dilatation as well as Disease of Valves*, 329. *Those peculiar to Disease of Valves*, 330; *viz.* (*a*) *unusual Severity*, 330; *and* (*b*) *Irregularity of the Heart's Action*, 331. *Pulse*, 331. *How affected by Contraction of the mitral Valve*, 331. *Difference between Intermittence, Irregularity, and Inequality explained*, 331. *How affected by Contraction of the aortic*

Valves, 332. *Pulse less irregular from Contraction on the right, than on the left Side of the Heart*, 333. (c) *Pain in the Region of the Heart a Sign*, 333. *Its Cause—either Over-tension or Inflammation*, 334. *Modes of Termination*, 334. *Sudden Death accounted for*, 335. *Prognosis to be cautious*, 335. PHYSICAL SIGNS. *Diagnosis of Disease of the Valves impossible before the Discovery of Auscultation*, 335. *Changes in the Signs resulting from the Substitution of the Author's View of the Heart's Action for that of Laennec*, 336. *The various Species of Murmur described*, 337. *Purring Tremor*, 338. *These applied as Signs of Disease of the Valves severally*, 340; *viz. of the aortic*, 340; *of the mitral*, 341; *of the aortic and mitral conjointly*, 342. *Of Regurgitation through the mitral*, 343. *Of the pulmonic Valves*, 343. *Of the tricuspid*, 343. *Fallacies and Means of avoiding them*, 344.

General signs of disease of the valves.—Whether the disease be cartilaginous, osseous, or consist of vegetations, the general symptoms are the same, if the degree of contraction be equal. Keeping in view the principles developed in the preceding section, I should assign to disease of the valves, as its general symptoms, first, a greatly aggravated form of the same as have already been assigned to dilatation of the ventricles; secondly, certain peculiar and distinctive signs which I shall presently describe.

1. Briefly to recapitulate these symptoms—they are, cough, copious watery expectoration, dyspnœa, orthopnœa, frightful dreams and starting from

sleep, œdema of the lungs, pulmonary apoplexy, passive hæmoptysis, (i. e. sputa stained with dark or grumous blood,) turgescence of the jugular veins, lividity of the face, anasarca, injection of almost all the mucous membranes, passive hæmorrhages, especially of the mucous membranes, engorgement of the liver, spleen, &c. and congestion of the brain with symptoms of oppression sometimes amounting to apoplexy.

The pulmonary symptoms result from engorgement of the pulmonary vessels, when the left valves are obstructed: and, where the obstruction is in the right valves, they result partly from engorgement of the bronchial veins, and partly from the quantity of blood transmitted into the lungs not being adequate to their demand—an unnatural state which gives rise to dyspnœa. (vid. Asthma.) In the latter case, hæmoptysis is more rare.

The symptoms affecting the system in general result from retardation of the blood in the venous system.

2. The peculiar and distinctive signs of valvular disease are, (*a*) that when the disease is combined with hypertrophy or dilatation, as is commonly the case, the symptoms are more severe than those of hypertrophy or of dilatation alone, the paroxysms of palpitation and dyspnœa in particular, being more violent, more obstinate, and more easily ex-

cited: (*b*) that the action of the heart is irregular. This, it is true, may sometimes be the case in hypertrophy and dilatation, but in them it is an accidental, not an essential character. The pulse, which we may regard as the representative of the heart's action, may in valvular disease, be small, weak, intermittent, irregular, and unequal; and it may even be small and weak while the heart is giving a violent impulse—a contrast which affords one of the strongest presumptions of valvular disease. The degree to which these characters of the pulse exist, depends on the situation of the valve contracted and the extent of its contraction. I shall therefore point out more particularly which of the characters are produced by given states of certain valves; for, this being known, we have a valuable means of ascertaining the situation and extent of the valvular disease.

The characters of the pulse just described, are most marked in contraction of the mitral valve; and, if its contraction be great, they are all invariably present; for as, in this case, the left ventricle is not freely supplied with blood, it is not stimulated to contract at the natural intervals and with suitable energy. The least degree of derangement is intermission; for in this the rhythm of the heart's action is not subverted, there being either a total omission of one or more ventricular contractions, or one, two or three feeble contractions audible by the stethoscope, but scarcely, if at all, sensible in the pulse; yet the next full contraction recurs at the correct interval: that is, supposing the pulse to beat crotchets, the contraction in question recurs at the crotchet. Irregularity is an ulterior degree of derangement, for here the rhythm *is* subverted, the beats recurring at irregular intervals. It is generally accompanied with inequality, some beats, both of the heart and pulse, being strong, and others weak.

A slight contraction of the mitral, when for instance the diameter of the aperture is not diminished more than a quarter of an inch, does not necessarily produce an unsteady pulse, as it still allows of an adequate supply of blood to the ventricle. When, however, the circulation is hurried, the pulse generally becomes unsteady.

Contraction of the aortic valves must be very great to render the pulse small, weak, intermittent and irregular. I have never seen it possess these characters in any marked degree unless the valves were either soldered together by cartilaginous degeneration, (Hedgley) or more or less fixed by ossification in the closed position, so that the aperture was only a limited chink.

An induration the size of an ordinary pea, has little effect on the fulness, firmness, and regularity of the pulse, and slighter degrees of contraction appear to have no effect on it whatever.*

The pulse is less irregular when the valvular contraction is on the right side than when on the left; the action of the heart appearing to be less under the influence of the right ventricle than of the left, in consequence, perhaps, of the muscular apparatus of the latter being stronger and more irritable. The pulse is not so small and weak from a contraction on the right side as on the left, and contraction of the tricuspid valve causes more irregularity than contraction of the valves of the pulmonary artery.

c. Pain in the region of the heart is another symptom of disease of the valves. It is true that palpitation may occasion pain though there be no disease of the valves, and I have frequently met with it from this cause in hypertrophy and dilatation. It is likewise true that palpitation may occasion pain though there be no disease of the heart whatever; I have often found it in hysterical females. But it is when the valves, the coronary arteries, or the commencement of the aorta, are indurated and inelastic, that pain occurs most frequently and with the greatest severity. Sometimes it is little more than an indescribable sense of obstruction or oppression in the præcordial region; but, in other cases, it is an intense lancinating or tearing pain, felt across the præcordia or scrobiculus cordis, (where it might be mistaken for inflammation of the stomach) and occasionally extending, with a sense of numbness, down the left arm to the elbow and sometimes to the fingers. Pain of this description has acquired the name of *angina pectoris*. (See ANGINA PECTORIS.)

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* Vid. Cases I. II. III. IV. IX. of aneurism of the aorta, by the author. Lond. Med. Gaz. Sept. 5th and 12th, 1829.

I believe this pain to be, in general, occasioned by the inelasticity of the ossified or otherwise indurated parts, which will not stretch equally with the other portions of the heart, when the organ is labouring under palpitation or engorgement. When inflammation of the interior of the heart exists, either alone or accompanying disease of the valves, it also occasions pain; but those authors have unquestionably been wrong who have considered inflammation the *sole* cause of pain, and have therefore assumed this symptom as proof of the inflammatory nature of disease of the valves.

The exact time and manner of the fatal termination in valvular disease, as in every other organic affection of the heart, is very uncertain. Sometimes the patient is reduced gradually to an extreme degree of emaciation and debility, and dissolution is duly announced by the usual premonitory symptoms. Sometimes he expires suddenly, after any trifling exertion or emotion, though the malady have made comparatively little inroad on the constitution. In this case the event must be attributed to the obstruction having attained, by the progress of the disease, such a point that the heart can no longer maintain the circulation against it. Why the heart should yield sooner in one case than another, can no more be explained than why one individual becomes tired by any effort sooner than another of apparently equal muscular force. Not unfrequently pressure on the brain, whether from serous effusion or venous congestion, is the immediate cause of death, and in this case coma gradually supervenes in the course of from three to four days or a week previous to the fatal event. (Dolan) In one case, under my care, of serous effusion, the patient suddenly uttered a shriek and fell at once into perfect coma. The same occurred in another case, in which there was only a small effusion of blood.

Hence, the prognosis must always be general as to time, and, if the case be considerably advanced, it must be guarded with a clause, that the patient is liable to die suddenly and unexpectedly.

Physical Signs.—Before the discovery of auscultation it was extremely difficult, and in many cases, utterly impossible to detect disease of the valves. Corvisart had the merit of discovering, as its signs, certain states of the pulse and a "peculiar vibration difficult to describe, sensible to the hand applied to the præcordial region:"—in other words the cat's *purring tremor (frémissement cataire)* of M. Laennec. But, as these signs may occur under other circumstances, they do not denote disease of the valves in particular, and are totally insufficient to indicate which is the valve affected. The accession of auscultation to the other means of diagnosis has rendered it possible to distinguish valvular disease, both in general and very often in particular, with almost complete certainty: a certainty, it may be remarked much greater than was supposed by the illustrious author of auscultation himself; for he did not give their full value to preternatural murmurs as signs of disease of the valves, in consequence of supposing that similar murmurs were produced by a spasmodic contraction of the muscular fibre of the heart and even of the arteries. I have elsewhere attempted to show that it is not the muscular contraction, but the movements of the blood, which are in all circumstances, the cause of these preternatural murmurs (vid. p. 47, and 56.) M. Laennec laboured under another disadvantage: he attributed the *second* sound of the heart to the auricular contraction; whereas, according to the experiments of the writer, it is referable to the ventricular diastole (vid. p. 26, and 36.) The substitution of this view of the heart's action for that of Laennec, fortunately does not falsify any of his physical signs, except that loudness of the second sound indicates dilatation of the auricles: it does not, to adduce a single instance, invalidate the fact that murmur of the second sound indicates disease of the auriculo-ventricular valve; but it affords a rational explanation of all the phenomena noticed by Laennec and renders various others available as signs, which to him were inexplicable and therefore useless.

Having explained (p. 56 et seq.) the mechanism by which preternatural murmurs are produced, I shall proceed to describe them as signs of valvular disease, according to the results of my own experience.

Bellows—filing—rasping—sawing—and musical or whistling-murmurs.—When a valve is contracted, the blood, in passing through it, is thrown into more than ordinary commotion and occasions a morbid murmur. This murmur has a *soft* character, like that of *bellows*, when the contraction has a smooth surface which does not greatly break the stream of blood, as when the morbid deposition consists of cartilage, fibro-cartilage or vegetations. But the murmur is rougher or more grating, like that of a *file* or *rasp*, when the deposition has a rugged, hard surface, as when it is osseous. Murmurs are more *hollow* when they are deep seated, as, for instance, in the auriculo-ventricular orifices; and more *hissing* or *whizzing* when they are superficial, as in the aortic orifice and, more especially, in the pulmonary orifice or artery (Cases of Collins and Wetherall), and the ascending aorta. The hollowness of the sound is referable to its remoteness and its reverberation through the chest. The *sawing* murmur is almost identical with the filing or rasping: the shade of difference can be better conceived than described.

The *musical bellows-murmur* is a perfect note like whistling or cooing. In the case of a patient who applied to me for " a noise in the chest" I heard it at the distance of two feet. In a case precisely similar which occurred to Dr. Elliotson, there was a very large and long vegetation in the mitral valve. I suspect, however, that the sound may possibly reside in the bronchial tubes, being created by the pressure of the heart during its systole; for I have several times heard it in the superior parts of the chest during the impulse of the heart, and then only. If this be true, it would be suspended, or, at least, modified, by holding the breath or by making a complete expiration—an experiment which it did not occur to me to try.

Purring tremor is another sign of disease of the valves. It arises from the vibrations into which the blood is thrown during its passage through an obstructed orifice, and is felt in the præcordial region and sometimes in the arteries. I have never felt it in the pulse when the patient was calm, unless hypertrophy was conjoined with obstruction of the aortic orifice or with roughness and dilatation of the ascending aorta: but I have felt it during palpitation, though there was neither hypertrophy nor disease of the heart or aorta. Whence I infer that it requires for its production in the pulse an increased force or velocity of the circulation, but not necessarily a valvular or aortic obstruction. In the præcordial region, it is more easily produced, and I have found it exist here when there was neither hypertrophy nor palpitation; but it was in cases in which the valvular obstruction was considerable, and the power of the heart certainly not deficient. It may be occasioned by obstruction not only of the semilunar, but also of the mitral and tricuspid valves, and in the latter cases it may accompany either the first or the second sound. When accompanying the first, it proceeds from regurgitation through the valve, and when accompanying the second, it results from the impeded passage of the blood from the auricle into the ventricle during the ventricular diastole. It rarely accompanies the second sound; because, as I conceive, the diastolic current is seldom strong enough to produce it. When from disease of the mitral valve, it is not felt in the pulse.

As purring tremor has the same origin as bellows and other murmurs, it often accompanies them; though, as it requires a greater degree of disease for its production, this is not always the case.

Having now given an account of the various murmurs and of purring tremor as signs of disease of the valves in general, I proceed to shew in what manner they constitute signs of disease of each of the valves in particular. It may be premised that, as the sounds of one side of the heart are audible on the other, the sound of the healthy side will partake of the murmur of the diseased side, and vice versâ.

Signs of disease of the aortic valves.—One of the murmurs above described is heard during the ventricular contraction about the middle of the sternum, (vid. p. 4) and is louder here than elsewhere. It is more or less hissing or whizzing, from being superficial, and it accordingly conveys the idea of being near to the ear. When a murmur of this kind is louder along the tract of the ascending aorta than opposite to the valves, and is, at the same time, peculiarly superficial and hissing, it proceeds from disease of that vessel. As the murmur often extends to the situation of the valves, it might easily lead to the supposition that they also are diseased, and it is sometimes very difficult to ascertain positively, that they are not. A murmur may accompany the second sound when there is regurgitation through the aortic valves, and its source may be known by its being louder and more superficial opposite to those valves than elsewhere. I have never found it strong, and I doubt whether it can be so, as the instantaneous manner in which the ventricle is re-filled by its diastole, must prevent the regurgitation from being considerable. Purring tremor generally requires for its production by disease of the aortic valves, a considerable degree of contraction of a rugged, hard nature, together with hypertrophy—especially, to produce it in the pulse.

Signs of disease of the mitral valve.—When this valve is contracted, the second sound loses, on the left side, its short, flat and clear character, and becomes a more or less prolonged bellows-murmur. When the valve is permanently patescent, admitting of regurgitation, the first sound likewise is attended with a murmur. (vid. p. 58—9.) These murmurs are louder opposite to the mitral valve (viz. at the left margin of the sternum, between the third and fourth ribs, i. e. about three or four inches above the point where the apex of the heart beats,) than elsewhere. They are also more hollow than murmurs of the aortic valves. By these two circumstances the murmur of the first sound may be known to proceed from the mitral, and not from the aortic valves: that of the second proceeds either from the mitral, or, what is much more rare, from regurgitation through the aortic valves. The means of ascertaining when the latter is its source have been explained above. With respect to the mitral, the murmur is diminished when the contraction of the orifice is extreme: when, for instance, the aperture does not exceed two or three lines in diameter; for then, the quantity of blood transmitted is not sufficient to create a loud murmur (vid. p. 57). For the same reason the murmur is diminished when the auricle is obstructed by a polypus; but the diminution under these circumstances, is of little importance to the diagnosis; as extreme contraction of the mitral, and polypi in the auricle, can be detected by various other pathognomonic signs elsewhere described (vid. p. 331, and the chapter on Polypi.)

Purring tremor may be produced by disease of the mitral valve, especially if the ventricle be hypertrophous and dilated, by which the currents through the valve are rendered stronger.

Signs of disease of the aortic and mitral valves conjointly.—The murmurs above described as characteristic of each, exist simultaneously in the situations of each. If the murmurs of the first sound be of a different *species* in the two situations—if, for instance, the murmur of the aortic valves be of the bellows kind, and that of the mitral resemble filing or rasping, it is still easier to determine that both valves are diseased.

Signs of regurgitation through the mitral valve.—These signs are, a murmur with the first sound, (vid. p. 59 and 61) louder in the situation of this valve than of the aortic, and a weak pulse, even though the impulse of the heart be violent. It is generally unsteady also. (Anderson and Dennis.)

Signs of disease of the pulmonic valves.—The signs are the same as those of disease of the aortic valves with this difference, that the murmur seems *close* to the ear, (vid. p. 4) and is equally hissing as in disease of the ascending aorta. Disease of the pulmonic valves is so rare that it ought never to be suspected unless the signs described are extremely well marked, or unless there be patescence of the foramen ovale, a state which experience has proved to be in general accompanied with contraction of the orifice in question. (Collins.)

Signs of disease of the tricuspid valve.—They are the same as those of disease of the mitral, except that the murmurs are loudest opposite to the valve in question: viz. at the middle part of the sternum, opposite to the inter-space between the third and fourth ribs, and a little to the right of the mesial line. As this valve is very seldom affected, the practitioner must be very cautious in pronouncing it diseased, especially as the pulse does not afford the same evidence as in contraction of the mitral orifice.

Such are the signs which, together with the general signs, are, so far as I can judge from my own experience, the best for the detection of disease of the valves. For several years they have never deceived me as to the general fact whether there was, or was not valvular obstruction: and they have seldom failed to indicate with perhaps more than necessary precision, the situation and nature of the affection.

To make the signs available, however, it is necessary to attend to several circumstances which might lead to deception. Bellows-murmur, as already fully explained, sometimes exists in the heart though there be no disease of the valves: namely, in nervous persons, (vid. p. 76) in cases of reaction from excessive loss of blood, (p. 76) of pericarditis and adhesion of the pericardium, (p. 75) and of hypertrophy with dilatation (p. 63). Murmur from these causes may easily be distinguished from that of valvular disease by the following criteria. When from nervous excitement, very common in hysterical females, it may be known by its being intermittent, ceasing when the nervous exacerbation subsides and the action of the heart becomes calm. When from reaction it subsides with the cessation of that phenomenon. When from pericarditis or adhesion of the pericardium, it may be known by the presence of signs of those affections. When from hypertrophy with dilatation, it may be known by its diminishing or ceasing when the action of the heart is calmed, as by repose, venesection, abstinence, &c. &c.

Contrasted with the above, the distinctive characters of valvular murmur are, that it is not universal over the heart, but confined in a great measure to the part corresponding with the valve affected; that it persists without intermission for an indefinite length of time, even though the heart be kept in a state of perfect calm; and that it is often of the filing, rasping, or sawing kind; whereas murmurs from other causes have almost always the softness of the bellows sound.

SECTION V.

ASTHMA FROM DISEASE OF THE HEART.

Disease of the Heart, as a Cause of Asthma, too much overlooked, 346. *Ancients considered all Asthmas purely nervous*, 346. *Error of this View*, 346. *All Asthmas have the same ultimate cause: viz. the Sensation of Want of Breath from inadequate Oxygenation of the Blood*, 347. *Three Causes of inadequate Oxygenation*, 347. *Varieties of Asthma according to their exciting Causes, compared*, 348. A. *Chronic dry Catarrh*, 348. B. *Pituitary Catarrh*, 349. C. *Mucous Catarrh*, 350. D. *Disease of the Heart*, 350. E. *Spasmodic Constriction of the Bron-*

chial Tubes, 351. *This exists in every variety of Asthma,* 352. *Conclusion that Asthma from Disease of the Heart is essentially the same as any other Asthma from an organic Cause,* 353. *This Asthma the most severe and fatal,* 354. *Error of attributing Asthma to Spasmodic Contraction of the external Muscles of Respiration,* 354. *Cases illustrating the contrary,* 355-6. *Cardiac Asthma imitates the other Varieties,* 358. *State of a Patient labouring under a Paroxysm of severe Cardiac Asthma,* 358. *Premonitory Symptoms of the Paroxysm,* 360. *Time of the Accession,* 361. *Paroxysm in Hypertrophy,* 363. *In valvular Disease and extreme Dilatation,* 363. *Subsidence of the Paroxysm,* 364.

ASTHMA has been too much regarded as independent of disease of the heart. Long treatises have even been written upon it without ever mentioning disease of this organ as one of its causes. It is, therefore, necessary to dwell a little on this subject, not only for the purpose of showing the magnitude of the error, but of making the reader acquainted with all the habitudes and aspects of a complaint, which is perhaps the most distressing in the whole catalogue of human maladies.

Corvisart has remarked that the ancients confounded under the name of *asthma,* and erroneously regarded as nervous affections, dyspnœas resulting from various organic diseases, particularly those of the heart and great vessels.

That they were wrong in regarding the latter as purely nervous affections, and especially in confounding them with those that were such, is unquestionable: but, whether they were also wrong in comprising them under the term *asthma*, is not so clear; for, on tracing back dyspnœa of that intensity and that character which amounts to asthma, (a dyspnœa which it is certain, both from the observation of the ancients and the concurrent testimony of all moderns conversant with diseases of the heart, may result from these diseases as well as from any other cause,) it will be found to originate primitively, in all cases, in the same ultimate cause, namely, inadequate oxygenation of the blood and the *sensation* of want of breath resulting from it. In order to render this apparent, it will be necessary to analyse the several varieties of asthma, and then compare them with each other.

It may be stated that inadequate oxygenation of the blood results in all ordinary cases, from one or more of three proximate causes: viz.

1. *Insufficient admission of air into the bronchial tubes and air-vesicles.*
2. *Insufficient admission of blood into the lungs.*
3. *Excess of blood in the lungs compressing the air-vessels, and preventing the free admission of air: also sometimes causing its own retardation.*

It will be found that to one or more of these causes all the varieties of dyspnœa and asthma are referable.

We shall, first, enumerate the varieties of asthma, and, secondly, examine how far they are severally connected with the causes in question.

I. All the varieties of asthma—to give an approximative statement probably very near the truth, are comprised under the following heads.

A. From *chronic dry catarrh*, and the *emphysema* resulting from it.

B. From *pituitary catarrh* (humoral asthma), whether acute or chronic, but more especially the latter, and the pulmonary *adema* resulting from it.

C. From *mucous catarrh*, especially chronic.

D. From *organic disease of the heart.*

E. From purely *spasmodic constriction of the bronchial tubes.*

I do not include amongst the varieties, one from compression of the lungs by hydrothorax, by tumours, by imperfect descent of the diaphragm, &c. because these rarely occasion what can strictly be called asthma.

II. A. *Chronic dry catarrh* is attended with intumescence and obscure or violet redness of the internal membrane of the bronchial tubes. The intumescence exists principally in the smaller tubes, which are sometimes completely obstructed by it; but it is also found in the larger. Andral has seen the bronchial trunk of a lung so contracted by this intumescence, that the air could scarcely enter; and in another case, the third and fourth bronchial divisions were contracted by the same cause.* Further, the tubes are more or less obstructed by an exceedingly viscous mucus often as dense as the vitreous humour of the eye, and when the dry catarrh is universal or even very extensive, it is almost invariably productive of emphysema.

It is manifest that this affection must be attended with an *insufficient admission of air* into the lungs. This, again, for reasons hereafter to be assigned, causes an *insufficient admission of blood.*

B. *Pituitary catarrh* is attended with moderate intumescence, slight softening, and partial redness of the pulmonary mucous membrane—a state intermediate between sanguineous and serous congestion, but partaking more of the latter. The quantity of phlegm expectorated, always considerable, is sometimes enormous, amounting to from four to six pints of thin glairy fluid in twenty-four hours.

The air passages being obstructed partly by the intumescence of their mucous membrane and partly by this fluid, it necessarily follows that there is an *insufficient admission of air* into the lungs.

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* Clinique Med. 2^e. partie, obs. ii. et iii.

C. *Mucous catarrh* is accompanied with more or less tumefaction of the bronchial membrane and obstruction of the calibre of the tubes. The expectoration, though less copious, and different in quality from that of pituitary catarrh, is, notwithstanding, frequently abundant, amounting to one or two pints or more in the day.

Hence there is an *insufficient ingress of air* into the lungs.

D. *Disease of the heart.* Sometimes blood does not enter the lungs in sufficient quantity, and this may arise either from the weakness of the right ventricle, from an obstruction in its mouth, or from increased resistance in the lungs, as, for instance, during sleep, when the respiration is less active. Hence results inadequate oxygenation of the blood, and its consequences the sensation of want of breath and dyspnœa. Cases exemplifying this will shortly be adduced. Meanwhile, it may be illustrated by a simple physiological experiment, viz. by making and sustaining a full expiration. This is attended, not only with a deficiency of air, but also of blood in the lungs, as is proved by the lividity of the face which ensues, by the elevation of the fontanel in infants, by the rise of blood in a tube inserted into the jugular vein, and, lastly, by the experiments p. 34, where it is demonstrated that, on suspending artificial respiration in a rabbit, the heart *instantly* became gorged, of a black colour, and distended to nearly double its natural size—a phenomenon which renders it sufficiently manifest that the blood, under the circumstances alluded to, does not freely enter the lungs. Now, the sensation of want of breath experienced on making a full expiration is familiar to every one, and it becomes intolerable if the expiration be long sustained.

Sometimes, on the other hand, blood exists in the lungs in excess, as is the case when the right ventricle is hypertrophous, or the left side of the heart obstructed, or, still more, when these two affections co-exist: also when the circulation is simply accelerated, as by palpitation, running, or by slighter effects in corpulent persons. Now, under all these circumstances, there is inadequate oxygenation of the blood; or, in other words, there is an excess of venous blood in the lungs: first, because the quantity of blood admitted exceeds its due proportion to the air in the organ; secondly, because the overloaded vessels do not always transmit the fluid with natural celerity; thirdly, because the engorgement of the mucous membrane on which the blood ramifies, constricts the bronchial passages, and prevents the free ingress of air, as proved by the feebleness of the respiratory murmur.

E. *Spasmodic constriction of the bronchial tubes* is presumed to exist, first, because, according to the researches of Reisseissen and others, the bronchial tubes appear to be provided with muscular fibres, and all muscles are liable to spasm: secondly, because asthma is found to occur without any organic cause (so far, at least, as our senses enable us to judge) sufficient to account for it: thirdly, because every form of organic disease above described may exist without causing dyspnœa of such intensity and of such a character as to constitute *asthma* properly so called. Thus, many have intense catarrhs and profuse expectoration without any asthmatic dyspnœa; and I have known a patient with a contraction of the mitral orifice to the size of a small pea and likewise with dilatation and softening of the heart and profuse expectoration, pass through a period of ten years to her grave, without ever experiencing a paroxysm of asthma, though a few steps across the room were sufficient to excite dyspnœa. (Mrs. —l—n.)

Hence, I apprehend that whatever be the organic cause of asthma, it requires for its production the super-addition of a state of the nervous system leading to spasmodic constriction of the bronchial tubes. In what this state consists, we can no more say than why one female falls into hysteria, and another does not, on seeing a third labouring under that affection; or why one lady " dies of a rose in aromatic pain," while another prefers it to all other perfumes.

Admitting that the constriction takes place, it is obvious that it will more or less close the bronchial tubes against the ingress of air; and this again, by preventing the free expansion of the lungs, will impede the influx of blood. Whence there is a double cause for the inadequate oxygenation of the latter, and, consequently, for the production and maintenance of the asthmatic paroxysm.

From all that has been said, we are now led to the resulting enquiry, what is the essential difference between asthma from disease of the heart and that from disease of the lungs. Putting aside that variety of asthma which, as not being attended with any *visible* organic derangement, (though it is, notwithstanding, highly probable that one exists,) may be regarded as mainly, if not wholly spasmodic, there does not appear to be any essential difference between the remaining varieties. Their organic causes are diversified, but they all ultimately produce the same effect, and it is the effect which constitutes the essence of the disease. This effect is, inadequate oxygenation of the blood, which causes the *sensation* of want of breath; and this when there exists more than what may be called mere dyspnœa, occasions spasmodic constriction of the bronchial tubes and its consequence asthma.

In reference to all the varieties of asthma, it may be remarked incidentally, that, whenever the disease, by preventing the ingress of blood into the lungs, leads to its accumulation in the heart, it may cause the enlargement of this organ.

We now proceed to the more particular consideration of asthma from disease of the heart.

This variety comprises by far the greatest number of the most severe and fatal cases of the disease. Some are of opinion that in other varieties the patient experiences an equal degree of suffering during the continuance of the paroxysm. I cannot say that this is consistent with my own observation. Though the same words may suit for the delineation of an attack of each variety, my feeling and conviction is, that I have never seen the patient suffer such intense and suffocative agony as in variety from organic disease of the heart.

Until the discovery of auscultation had in some degree dissipated the deep obscurity of the affections of this organ, the fact that they were a cause of asthma was scarcely known; and even at the present day, there are few errors more common than that of attributing asthma to other causes when it originates solely in the heart. For instance, a theory of this description which has of late years been more widely disseminated than perhaps any other, consists in ascribing asthma to a spasmodic or convulsive contraction of the external muscles of respiration, much dependent on habit.

The action of these muscles, so far from being morbid or dependent on habit, is a natural instinctive and salutary effort to prevent suffocation, the stimulus to which consists in an exaggeration of that which excites the muscles in ordinary respiration—namely, as above explained, the *sensation* of want of breath, from inadequate oxygenation of the blood. Nothing is more common, for instance, than to see a patient with diseased heart, while sleeping tranquilly, start up and begin to respire with violence. Here it is obvious that the necessity for violent respiration preceded the act; and the necessity depends on impeded transmission of blood through the heart and lungs; for starting is invariably accompanied by palpitation and preceded by frightful dreams or some sensation of præcordial distress, indicating an obstructed circulation. I have frequently examined the heart and lungs by auscultation immediately before the supervention of a paroxysm of dyspnœa, and have always found that the heart began either to palpitate, or to act in that irregular, confused and, as it were, struggling manner, which denotes its engorgement. I was therefore enabled to tell the patient that difficulty of breathing was coming on, to which, with some astonishment, he would reply in the affirmative, being himself forewarned of the approaching accession by a feeling of anxiety and straitness in the præcordia. This fact is so universally true that any one may satisfy himself of it by entering an hospital and gently placing a patient with orthopnœa from disease of the heart, in a rather uneasy position, when the series of phenomena described will become manifest.

Dr. Burrows communicated to me the particulars of a case, recently under his observation, in which the respiration was alternately violent and tranquil under the following circumstances. The patient dozed for a few minutes at a time, during which his complexion became livid and his pulse more and more feeble, oppressed and irregular. He then started up, and after a few violent wheezing respirations, relapsed into the same calm doze. In this case the mitral orifice was contracted to the size of a pea. Now, there can be little doubt that, as the sensation of want of breath is less felt, and the muscles of respiration are, consequently, less stimulated by it, during sleep,—in simple language, as the respiration is more feeble during that state;* it did, in the present case, keep the lungs sufficiently expanded to maintain an adequate circulation through them; whence ensued engorgement of the heart and venous system of the body, with insufficient arterialization of blood in the lungs, and the sensation of necessity for breathing resulting from it, which series of phenomena was relieved by the succeeding violent respirations.

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* As the respirations are slower, they might be supposed deeper: but, with the exception of those deep inspirations that are taken occasionally during, and more particularly at the *breaks* of sleep, the expansion of the lungs during sleep will be found by auscultation to be less than at other times.

I have frequently observed this series of phenomena in a greater or less degree. In one case violent gasping and wheezing respiration, lasting from a few seconds to two or three minutes, occurred at intervals of four or five minutes, during which the patient dozed, even though sitting erect on a stool and undergoing a stethoscopic examination; and this series of actions continued so long as the patient remained disposed to sleep in that situation.*

In all these cases, it is manifest, that the action of the muscles of respiration was consecutive to the obstruction of the circulation, and that it was not dependent on any spasm of those muscles, but simply on the necessity for breathing, which instinctively exerted them to a salutary preservative effort.

Asthma from disease of the heart often imitates the characters of the other varieties; and this perhaps for a very simple reason; that the lungs are in much the same state as in those varieties. Thus, it is *humid* when there is permanent engorgement of the lungs, causing copious sero-mucous effusion into the air vessels, as from contraction of the mitral valve. It is *dry*, when the engorgement is only temporary, as in cases of pure hypertrophy. It is *continued* when there is a permanent obstruction to the circulation; and any of the varieties may be *convulsive* when the heart has sufficient power to palpitate violently. The worst cases of convulsive asthma from disease of the heart are those of hypertrophy with dilatation and a valvular or aortic obstruction.

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* Case of Lindsay Corstairs, Lond. Med. Gaz. Sept. 5, 1829, p. 417.

We shall now examine the state of a patient labouring under severe asthma from disease of the heart, and then take a more strictly medical view of the nature and progress of the asthmatic paroxysm.

The respiration, always short, becomes hurried and laborious on the slightest exertion or mental emotion. The effort of ascending a staircase is peculiarly distressing. The patient stops abruptly, grasps at the first object that presents itself, and fixing the upper extremities in order to afford a fulcrum for the muscles of respiration, gasps with an aspect of extreme distress.

Incapable of lying down, he is seen for weeks, and even for months together, either reclining in the semi-erect posture supported by pillows, or sitting with the trunk bent forwards and the elbows or fore-arms resting on the drawn up knees. The latter position he assumes when attacked by a paroxysm of dyspnœa—sometimes, however, extending the arms against the bed on either side, to afford a firmer fulcrum for the muscles of respiration. With eyes widely expanded and starting, eye-brows raised, nostrils dilated, and the head thrown back at every inspiration, ghastly and haggard he casts round a hurried, distracted look of horror, of anguish, and of supplication; now imploring, in plaintive moans, quick, broken accents, and stifled voice, the assistance already often lavished in vain; now upbraiding the impotency of medicine; and now, in an agony of despair, drooping his head on his chest and muttering a fervent invocation for death to put a period to his sufferings. For a few hours—perhaps only for a few minutes—he tastes an interval of delicious respite, which cheers him with the hope that the worst is over and that his recovery is at hand. Soon that hope vanishes. From a slumber fraught with the horrors of a hideous dream, he starts up with a wild exclamation that " it is returning." At length, after reiterated recurrences of the same attacks, the muscles of respiration, subdued by efforts of which the instinct of self-preservation alone renders them capable, participate in the general exhaustion and refuse to perform their function. The patient gasps, sinks, and expires.

Such are the sufferings of an asthmatic from disease of the heart. We have now to take a more strictly medical view of the nature and progress of the asthmatic paroxysm.

If about to be severe, it is generally preceded by certain premonitory symptoms, which, though not so marked as in ordinary asthma, are much of the same nature—probably because derangement of the circulation and imperfect oxygenation of the blood are present in both. In cardiac, however, many of the nervous symptoms are often deficient which characterize the ordinary varieties. One of the most common and efficient exciting causes of cardiac, as of all other asthmas, is, derangement of the stomach, the irritation of which extends to the heart, and stimulates it to inordinate action. The irritation, according to the theory of Sir Charles Bell, is propagated through the medium of the par vagum, by which nerves the stomach and heart are closely associated as parts of the respiratory system. Accordingly, after a feeling of acidity, flatulence, or a load on the stomach from undigested food, often accompanied with abdominal distention, the patient experiences pain, weight, and constriction in the forehead and over the eyes accompanied (if the case be one of hypertrophy of the left ventricle) with throbbing of the temples and the sound of rushing waters. He feels a sensation, scarcely to be defined, of oppression, tightness and anxiety about the præcordia, sometimes with slight palpitation. A gentleman at present under my care presents the above symptoms habitually and in the most marked manner. Sometimes the patient is drowsy, listless, restless, irritable, and impatient not only of society but of the attentions of friends: these symptoms, however, are, in general, more prevalent in ordinary asthma. The signs described afford the experienced asthmatic well-known assurance of the approaching attack.

They gradually become worse and worse, especially after a meal, and eventually burst into a paroxysm. The time of the accession is less regular than in ordinary asthma, being more dependent on the state of the heart, which is liable to accidental excitement at any moment from a variety of causes. The fit, however, as in ordinary asthma, is, on the whole, more apt to supervene during the evening or early part of the night; and this, as appears to me, for two reasons: 1st. The recumbent position is unfavourable to respiration, the diaphragm being pressed upwards by the abdominal viscera, and the expansion of the chest being opposed by its own weight: 2d. During sleep respiration is not assisted by the will, which, during the wakeful state, from the sensation of want of breath being more acutely felt, is ever ready to maintain the body in the position most favourable to breathing. From the co-operation of these two causes, therefore, the circulation becomes so far embarrassed before the patient is aroused to a sense of his condition, that it can only be relieved by those violent efforts which constitute the asthmatic paroxysm. He accordingly awakes, generally with a start, in a fit of dyspnœa, accompanied either with violent palpitation, or a distressing sense of anxiety in the præcordia and great constriction of the chest, as if it were tightly bound. He is compelled to assume a more erect posture, and intensely desires fresh, cool air; the respiration is wheezing and performed with violent efforts of all the muscles of respiration both ordinary and auxiliary. The inspirations are high and accompanied with apparently little descent of the diaphragm, and the expirations are short and imperfect. The surface is chilly, the extremities are cold, and the face is pale and sometimes livid.

In cases in which the pulmonary congestion is only *temporary*, as in hypertrophy either simple or with dilatation, there is no cough beyond a few slight and ineffectual efforts, producing little or no expectoration; and in such cases the fit subsides as soon as the engorgement of the heart and great vessels is relieved, which nature generally effects in two or three hours or less, by determining the blood to the surface and creating diaphoresis. In some instances I have known this to be regularly accompanied with a copious secretion of pale urine and a purging alvine evacuation. (Case of May.) In this case the attacks recurred, according to the assertion of the patient, every night for several years.

The pulse, though at first full strong and bounding, may, during the worst of the paroxysm, become feeble and small, and the sound and impulse of the heart may be diminished; and this in cases even in hypertrophy; for the organ, being gorged to excess, is incapable of adequately contracting on its contents.

Such is the nature of an asthmatic fit when the pulmonary congestion is only temporary: the case is different when it is *permanent*, as in valvular disease and in some extreme cases of dilatation. For then, there is violent cough in suffocative paroxysms, accompanied, at first, with difficult and scanty expectoration of viscid mucus, but ending gradually in a copious and free discharge of thin, transparent, frothy fluid, occasionally intermixed with blood. This evacuation, by disgorging the pulmonary capillaries, affords great relief to the cough and dyspnœa. As, however, the transudation of the matter to be expectorated into the air-passages, and its final elimination, are slow processes, paroxysms of this description are much more protracted than those of dry asthma from hypertrophy. They frequently last five or six hours, and I have known them persist, with only occasional remissions, for two, three or more days. During the attack, the pulse is quick, small, and weak, often irregular and intermittent. The slowness which the latter characters sometimes appear to give it, has led some authors to suppose that the circulation through the heart is little disturbed in asthma. This is in some degree true in reference to other varieties of asthma; but it is always incorrect in reference to that from disease of the heart.

As the paroxysm subsides, the anxiety and constriction decrease, the respiration becomes less frequent, high, and laborious, and the pulse becomes slower, fuller, and more regular. But some degree of wheezing and tightness of the chest remain, and the paroxysm is very apt to return for two or three nights successively, and sometimes for a much longer period, until the lungs are freely unloaded by copious expectoration. It may, indeed, continue to recur at brief intervals for an indefinite period, or the patient may never be wholly exempt from some degree of asthmatic dyspnœa.

A severe asthmatic attack from disease of the heart is in general far more injurious in its consequences than one from a mere affection of the lungs.

SECTION VI.

TREATMENT OF VALVULAR DISEASE.

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According to the foregoing principles, the exciting causes of valvular disease are, 1. over-tension of the valves by the force of the circulation, and, 2. inflammation,—generally of the chronic kind. If it were possible to ascertain that these causes were in operation, before they had actually occasioned an organic change, it would most probably be possible, in many cases at least, to counteract their effects and prevent the formation of the disease. But, unfortunately, there are no positive signs of the latent mischief but what result from the disease already formed—from the obstruction itself: and, as, in the present state of our knowledge, we are not acquainted with any means of *removing* a valvular obstruction, the indications of treatment in this disease are, to prevent its increase, to counteract its tendency to induce hypertrophy and dilatation, and to relieve the symptoms of an obstructed circulation.

The remedies calculated to answer these indications, are, in general terms, such as diminish the force and activity of the circulation: namely, occasional venesection to a moderate extent, an unstimulating and rather spare, though sufficiently nutricious diet, a tranquil life with respect both to the body and the mind, and a good state of the digestive organs and alimentary canal.

If there be distinct signs of inflammation of the valves, to the above remedies may be added cupping or leeching on the præcordial region, with counter-irritants as blisters, setons, issues and the tartrate of antimony in the form either of ointment, or plaster with the empl. picis comp. and bee's wax. I have also found digitalis very useful under these circumstances.

The extent to which any remedy must be carried can only be determined by the particular circumstances of each case. If, for instance, the patient be robust and plethoric, depletory measures may be pursued to a greater extent, and vice versâ. In general, if the valvular obstruction is not very considerable and there is no hypertrophy or dilatation, and no tendency to plethora, an abstemious, light diet, and a scrupulously tranquil life with an open state of the bowels, constitute all the prophylactic treatment that is necessary; and it is satisfactory to know that, by these means, danger may in many instances be completely averted. I have several times known patients with a moderate—even with a rather considerable valvular obstruction, attain the age of sixty, seventy, and even eighty, though the symptoms, judging from their account, had commenced early in life.

On the other hand, if precautionary measures be neglected and hypertrophy or dilatation superinduced, there is no organic disease of the heart, except adhesion of the pericardium, which tends more rapidly to its fatal termination. Hence the great importance of detecting and attending to disease of the valves in its earliest stage.

When the obstruction has become very considerable, has produced hypertrophy or dilatation, and is attended with much dyspnœa, orthopnœa and dropsy, the case is one of the most difficult that the practitioner can encounter. The most urgent symptoms, however, generally admit of being removed for a time; and the amelioration which takes place is sometimes truly astonishing. But, unhappily, the complaint seldom fails to return with greater or less promptitude. If the patient be youthful and of a robust constitution, the relapse may not occur for several months, especially if he have not been affected with dropsy, or only for the first time; but if he be of a shattered constitution and have previously had severe attacks, the symptoms commonly return the moment he resumes any active occupations. In an ulterior degree of the disease, no sooner are the symptoms dispersed than they return, though the patient has not been guilty of any indiscretion.

When this is the case, the fatal event is never far remote, and may be expected to occur at any moment.

The remedies suitable for the treatment of the cases described, are, abstractions of blood, purgatives, diuretics, sedatives, revulsives, a spare diet and, what is paramount in importance to all, complete repose. These remedies, however, are not to be employed at random: so used, they might not only be unavailing, but directly destructive. It is only by adapting them to the character of the organic cause of the disease—only, in short, by a sound diagnosis, that they can be administered safely and effectually. It is necessary, therefore, to enter into further particulars relative to their nature and mode of application, and this may be most conveniently done by adverting separately to each.

Blood-letting.—When, with the valvular obstruction, there is hypertrophy or hypertrophy with dilatation, bleeding is generally necessary and may be repeated in small quantities, two, three or more times, according to the strength of the patient and the urgency of the palpitation and dyspnœa. Some have recommended that blood-letting be practised in valvular disease in the unsparing manner of Albertini and Valsalva. The results of my own experience lead me to dissent entirely from this doctrine. Excessive bleeding cannot remove the valvular obstruction—cannot, therefore, *cure* the disease; consequently its employment with this object is inappropriate. It is, moreover, directly injurious; as it reduces the patient to a state of debility which renders his circulation more liable to be embarrassed by the valvular obstruction, and his constitution more susceptible of the inroads of the malady. I have always observed blood-letting to be most serviceable in valvular disease when carried only just so far as to relieve the existing urgent symptoms without encroaching on the constitutional powers. During the intervals of the attacks it is advantageous to draw from four to eight ounces of blood occasionally, whenever dyspnœa becomes urgent and the heart's impulse more than ordinarily strong.

If, instead of hypertrophy, dilatation, either simple or attenuated, be conjoined with valvular disease, blood-letting is less necessary, and is more injurious if carried to excess. It should be resorted to reluctantly; only when imperiously demanded by excessive dyspnœa which other means have failed to relieve; the least quantity that suffices to afford relief should be drawn; and the depletion should not be repeated if it can possibly be avoided. Attention to these rules is still more necessary in the aged. The greater the valvular obstruction, the greater is likely to be the embarrassment of the circulation, if the power of the heart and the system be reduced below a certain point. Of this I feel satisfied from reiterated observations.

Diuretics.—When there is dropsy and a scanty secretion of high coloured urine, remedies of this class are of the greatest utility. In most cases, indeed, the dyspnœa, palpitation, cough, &c. decrease in the same proportion as the urine increases and the dropsy disappears. Nor is it only when dropsy has actually appeared that diuretics are useful. They are remarkably beneficial in any stage of the disease; for, by drawing off the serous portion of the blood, they diminish the quantity, without deteriorating the quality of that fluid, and thus relieve palpitation and dyspnœa and obviate infiltration, without materially reducing the patient.

Diuretics are very variable in their effect, a weaker sometimes answering perfectly after a stronger has failed. When, therefore, one does not speedily produce the effect, another should be tried. The surest way is to employ several at once. A pill consisting of three grains of blue pill, one of pulv. scillæ, and one or half of one of pulv. digitalis, given three or four times a day, seldom fails: or it may be given once or twice a day with a draught of Tr. scillæ. mxx. Sp. ætheris. nit. and Sp. Junip. C. comp. a̅a̅n. ʒss. ad ʒi, in Dec. Spartii. ℥iss. twice or thrice a day. I have sometimes found all these fail until ʒij or ʒiij of infusion of digitalis was added to the draught. Its effect, however, must be carefully watched. Supertartrate of potass is always a valuable auxiliary, and may be given to the extent of ʒij or ʒiij in twenty-four hours, either in the form of a drink, of electuary, or in the above draughts.

Sometimes diuretics cannot be made to produce any effect: it is then necessary to resort to purgatives, as will presently be explained.

In very feeble and reduced patients, dropsy should not be too rapidly evacuated; as the process is attended with a degree of exhaustion, which is often fatal. The period, indeed, immediately succeeding the disappearance of dropsy is, on this account, one of the most critical. The older physicians were aware of this, and ascribed it to the accumulation of fluid in the internal cavities. Such, however, is not always the cause; for, in cases that terminated fatally at the period alluded to, I have frequently ascertained, both by auscultation, percussion and post-mortem examination, that the internal and external dropsy disappeared simultaneously.

Purgatives.—When diuretics do not remove dropsy, purgatives will frequently produce that effect. The two classes of remedies may, indeed, be combined with great advantage, when the patient is strong enough to bear them. The drastic hydrogogue purgatives are the most efficacious, as tinct. jalapæ, elaterium, &c. The effects of the latter are sometimes truly astonishing. I have seen an extreme universal anasarca removed by it in three or four days. The remedy is apt, however, to be excessively violent in its operation and should therefore, only be given to strong subjects. As its effect varies in different individuals, it should be tried at first in small doses, as from one eighth to one fourth of a grain. With caution it may be carried to two grains. I generally give it in the form of pills with pulv. capsici, which obviates its griping effect; sometimes I add a grain or two of calomel. A single pill should produce six or eight watery evacuations, and it may be given two or three mornings successively, or every second or third morning, according to the strength of the patient. All the other purgatives may be useful, especially such as produce watery evacuations. A very good one is, the infusion of senna, with tinct. jalapæ ʒi. and tartrat. or acetat. potass ʒij.

An occasional purgative is sometimes very beneficial though there be no dropsy; as, for instance, when an asthmatic attack has appeared to be induced by an excess of bile, by undigested food, or by acrid or long detained fœces in the intestines. Under such circumstances a purgative often alleviates, and sometimes terminates the attack. Except with a view of removing dropsy, or plethora in cases where hypertrophy is conjoined with valvular disease, frequent, systematic purging should be avoided on the same principle as blood-letting: viz. lest it should too much reduce the system.

Diaphoretics.—When there is anasarca, cutaneous transpiration contributes very powerfully to remove it. A lady at present under my care and subject to frequent attacks of anasarca, often finds the swelling disappear in twenty-four hours with copious perspiration. Strong stimulating sudorifics, however, should be avoided, as they are both too debilitating and too exciting to the circulation. Gentle saline diaphoretics are the best, and their effect may be promoted by warm clothing and the occasional use of the warm bath to keep the skin soft and open. When there is no anasarca, and no *permanent* pulmonary engorgement with expectoration, diaphoretics, beyond warm clothing, are of little use, except occasionally, to relieve asthmatic attacks. For the latter purpose I have generally found them of great utility; but, as internal remedies of this class are slow in their operation, they should be assisted by fomenting the hands and feet, or immersing them in warm water, at the same time keeping the trunk covered. If perspiration can thus be gently elicited without heating and stimulating the patient, it is one of the most effectual means of curtailing a paroxysm. Nature herself indicates the remedy; as an asthmatic paroxysm often terminates with spontaneous diaphoresis. In one patient under my observation (May) this occurred nightly and to an extreme degree for several years.

Emetics.—Are extremely useful or extremely pernicious according as they are judiciously given, or the reverse; and it is only by a sound diagnosis that the practitioner is enabled to judge whether they can be safely administered or not. When there is an undigested, bilious or acid load on the stomach, exciting the asthmatic fit, its removal by an emetic often affords instantaneous relief. But the medicine should be one which simply evacuates the stomach without much shaking the system, as ipecacuan, with sulphate of copper or of zinc, but by no means tartrate of antimony.

If the disease of the heart and the embarrassment of the circulation be great, even such an emetic cannot be given without danger of aggravating all the symptoms, I have seen emetics, administered under these circumstances, exasperate and prolong the paroxysm, increase the frequency of its recurrence, and speedily bring the patient to his grave. They may even cause death during the paroxysm. Their dangerous effect consists in their increasing the engorgement of the heart and the obstruction of the circulation. For this reason they should not be ventured upon in disease of the heart simply for the object of promoting expectoration—an object which may by other means be much more safely and effectually accomplished. In other varieties of asthma, especially that from pituitary catarrh, they are peculiarly beneficial by promoting the expectoration of the immense accumulations which take place in the lungs. Hence the importance of carefully distinguishing between these two classes of cases.

I have said thus much respecting emetics, because they have been alternately both extolled and decried, the parties using them under different circumstances, and neither perfectly understanding on what their good or bad effect depended.

Though emetics are objectionable except for the purpose of evacuating the stomach, small doses of ipecacuan or tartrate of antimony are useful as diaphoretics and expectorants. When the obstruction of the circulation is great, they cannot safely be carried to nausea, as this state is apt to bring on a langour of the circulation which leads to the formation of polypi in the heart. In the case of a lady lately under my care, nausea came on unexpectedly at the moment when she had just been relieved of an excessive dropsy: it was followed by suffocating dyspnœa, an imperceptible pulse and other symptoms indicating the formation of a polypus in the heart. She died in a week and the polypus was found.

Puncturing.—When dropsy has failed to be relieved by other means, and the cutaneous tension has become intolerable, the practitioner is compelled to resort to puncturing. I say compelled, because the remedy is a last and dangerous resource. The danger, however, may be considerably diminished by making small punctures with a grooved needle and allowing the fluid to ooze *slowly* during four or five days or a week. When *incisions* are made with a lancet or scalpel, and the fluid is evacuated quickly, as in twelve to forty hours, the patient, according to my observation, generally dies. This event sometimes results from sloughing of the incisions, but more commonly from exhaustion induced by the *sudden* evacuation of the fluid. In one instance I saw the patient die from hæmorrhage.

Setons, issues, and blisters on the præcordial region, are of no use unless there be chronic inflammation of the heart: the pain and irritation that they occasion are often injurious.

Expectorants.—When there is permanent engorgement of the lungs, free expectoration always affords relief, and I have seen great dyspnœa result from its suppression by an incipient catarrh, a dry sharp atmosphere, and even a dose of laudanum. Most asthmatic fits dependent on valvular obstruction, terminate with copious expectoration of thin sero-mucous fluid. This secretion, therefore, should always be maintained when there is a tendency to it.

As the stomach in disease of the heart is extremely fastidious and delicate, oily, sweet, and nauseous expectorants should be carefully avoided. Squill with an acid, as the acetic or nitric, has been found by experience to be the most efficacious remedy of this class. Vinegar of squill has been highly extolled by Floyer, and tinct. scillæ, g^{tt} x.—acid nitrici, g^{tt} vi—extr. hyoscyami, gr. iij—aquæ puræ, ℥iss, as a draught every three or four hours during the paroxysm, is the favourite prescription of Dr. Bree for the asthmatic paroxysm of his *first species*, i. e. "from effused serum in the lungs." Mixt. ammoniaci, though in general too heating for the young, is a useful expectorant for the old, when sufficiently diluted. The same may be said of the decoction of seneka. When asthma, in middle-aged and elderly persons, takes the character of peripneumonia notha in the winter and spring, seneka, according to Dr. Bree, is the most certainly useful remedy that he has tried. When, in such cases there is fever, the seneka should be combined with liq. acetatis ammoniæ; and when the fever subsides, the addition of squill and Tr. camphoræ comp. powerfully promotes expectoration, perspiration and the secretion of urine. Ipecacuan and tartrate of antimony in small doses, are valuable expectorants as well as diaphoretics. They may be carried to a slight degree of nausea if the obstruction of the circulation is not very great.

Phlegm accumulates during sleep, and it is for this reason principally, that the patient suffers more on first rising in the morning. The elimination of the phlegm is greatly facilitated by a cup of any hot fluid, especially coffee; and, to allay the nervous irritability of the lungs which generally leads to coughing before the phlegm is sufficiently detached to be thrown off with ease, I have found from half a drachm to a drachm of tinct. camphoræ comp. of great utility.

Expectorants should not be constantly given, but only to relieve an asthmatic paroxysm or to restore the pulmonary secretion when accidentally suppressed.

Gases.—The effects of atmosphere on asthmatics are so diversified that they can scarcely be reduced to any general rule. When, however, expectoration is habitually copious, a moist warm atmosphere favours it, probably by relaxing the pulmonary vessels. A clear, sharp air, on the contrary, checks it and thus increases dyspnœa. Again, such an air relieves dyspnœa when it depends, not on engorgement of the lungs, but on a languid action of the heart, as in dilatation with attenuation; and this it does by stimulating and bracing the system and causing a freer circulation through the lungs and more perfect arterialization of the blood. Electricity appears to act in the same way when it produces any good effect. I have never tried the inhalation of oxygen, but it is highly commended by Dr. Beddoes and others; and it is rational to think that, in suffocative dyspnœa from retardation of the blood in the lungs, it would relieve the anxiety and straitness by causing a more perfect arterialization.

Smoking tobacco or stramonium sometimes affords extraordinary relief to asthmatics, and this it does partly, perhaps, by increasing the bronchial and salivary secretion, but more especially by its sedative and antispasmodic effect in tranquillizing the nervous system, resolving the bronchial spasm, and allaying the sensation of want of breath. The experience of the patient is the only certain criterion of its utility. In many cases I have certainly seen it prejudicial. Its utility is the greatest in those who are of a highly nervous irritable habit, and in whom asthma displays most of the spasmodic character.

Antispasmodics.—While the Cullenian doctrine, that spasmodic constriction of the bronchi was the sole cause of asthma, prevailed, remedies of this class were much in vogue, but experience has not realised the high expectations to which the theory gave rise. Antispasmodics are useful auxiliaries, but cannot be depended upon alone. When they contribute to diffuse and equalise the circulation in disease of the heart, they are beneficial: when they fail to produce this effect, they are of little use. In an incipient paroxysm from slight disease of the heart, I have frequently found a draught of sp. ammoniæ aromat. or fœtid, with æther and laudanum, promptly restore the colour to the face and warmth with perspiration to the skin, with general relief. In one case of hypertrophy with dilatation and adhesion of the pericardium, a glass of gin and water had always the effect. Sometimes gr. x to xv of carbonate of ammonia is more efficacious than any other remedy. The solution of assafœtida has also appeared to me to be very powerful, but few patients can be prevailed upon to take it.

In most instances, the antispasmodic, whatever it be, is productive of eructation, and to this, in some measure, I attribute its beneficial effect, as flatulence alone suffices to occasion a paroxysm. The eructation sometimes occasioned by the remedies themselves, especially æther, must not be mistaken for the extrication of real flatus.

When the paroxysm is fully established and results from a great degree of organic disease of the heart, antispasmodics have little or no effect in affording relief, and large doses of sedatives, as opium, hyoscyamus or conium, or of stimulants as æther, often prolong it. In conjunction with other means, however, moderate doses may be tried, and, if the patient feel himself relieved, they may be continued, and vice versâ.

Digitalis, according to my experience, is an excellent adjunct to an antispasmodic draught: g^{tt} xx or xxx of the tincture may be given every three or four hours, with g^{tt} vi to x of tinct. opii, or, if that disagree, of hyoscyamus, in cinnamon water. Care should be taken to intermit the digitalis before its specific poisonous effect is produced.

In suffocative, agonizing orthopnœa, when the restlessness and jactitation of the patient, aggravates the distress, I have often found narcotics afford great relief simply by inducing sleep and a diminished sensation of suffering, and they should always, I think, be used under these circumstances, to procure the patient a remission when the fatal event is close impending.

Stomachics.—The correction of dyspepsia is of the first importance in organic disease of the heart; as the fit is often dependent upon it alone. Two gentlemen at present under my care for hypertrophy with dilatation, never suffer palpitation, dyspnœa, or headache except when affected with acidity, flatulence, &c. Such cases are often mistaken for "*the stomach*" alone—a most dangerous mistake: of the individuals alluded to, one has had a fit of apoplexy, and the other has been repeatedly rescued from it by prompt cupping. When there is acidity, antacids, of which chalk is the best, should be freely given every third or fourth hour, its constipating effect being counteracted by the previous or simultaneous exhibition of a few grains of rhubarb. I have already stated that the stomach, if loaded, should, in the first instance, be evacuated by a gentle emetic, copious draughts of tepid water or chamomile tea being taken to ensure its full effect. This treatment will generally terminate an attack dependent on dyspepsia, in two or three days. Towards the close of the attack, sedatives, as opium or hyoscyamus, assist by tranquillizing the nervous system.

Not only antacids, but also acids themselves, have been proved by experience to correct acrimony of the stomach accompanied with flatulence and distention. Their efficacy is the greatest when the acrimony is bilious, and they then act, in all probability, both by neutralizing the alkaline qualities of the bile, and exciting the stomach to an altered and more healthy secretion. That they possess the latter property, is to be inferred from their correcting acidity and preventing fermentation even when there is no bile. A sour apple is a popular remedy for heart-burn. The acids to be employed, are, the mineral acids much diluted and also the acetous. Saccharine acids, as oxymel, acescent fruits, raspberry vinegar, &c. should be avoided, as they are apt to be more injurious by their fermentation, than beneficial by their acid qualities. Dr. Bree has found vinegar extremely useful in the paroxysm of his first species "from effused serum in the lungs." In the access of the fit he unites it with squill or ipecacuan, to produce vomiting. Afterwards, according to the progress of the complaint and the violence of the spasms, æther may be added, and also opium, if necessary.

To give tone to the stomach, bitters are very useful. Infusions should be employed during the paroxysm, as tinctures are too stimulating; but after the second or third day, when the patient begins to amend, either the one or the other may be used. The bitters may be conjoined with the antacids, acids, &c. Griffith's mixture is very beneficial in debilitated subjects, in the intervals between the fits.

Tonics.—When disease of the heart is of the hypertrophic kind with increased activity of the circulation, tonics are obviously inappropriate: when it is of the dilated kind, with languor of the circulation and atony of the system, they are remedies of the greatest value, and it is by them only that a complete cure can be effected. All the tonics, of which the preparations of iron are the best, may be used according to the discretion of the practitioner. Of the advantages of bracing air and exercise and of the shower bath, I have spoken in the article on dilatation. A discreet use of the cold bath also is highly beneficial.

Such are the remedies to be used in the treatment of organic disease of the heart. It cannot be too strongly inculcated on the practitioner, that the disease, when remediable, is not to be cured by *relieving* the paroxysm, but by *preventing* its occurrence. Every attack gives the patient much ground to retrace: a single attack may undo the progress of a year, and death may result from the indiscretion of a day. Great firmness is necessary on the part of the physician to impress this strongly on the mind of the patient; for the latter, when his *feelings* are easy, can seldom—very seldom—be made to comprehend that the necessity for his rigid adherence to medical, regiminal, and dietetic discipline is equally imperative.

The practitioner, however, is not the less to study the means of relieving the paroxysm; not only because in it he has perhaps the greatest of human sufferings to alleviate, but because by curtailing the attack he increases the chances of a cure.

CHAPTER X.

ANEURISM OF THE AORTA.

SECTION I.

CLASSIFICATION, NOMENCLATURE, ANATOMICAL CHARACTERS AND FORMATION OF ANEURISMS OF THE AORTA.

Four Species and their Names, 387. Dilatation, *Formation,* 387. *Anatomy,* 388. *Coagula in,* 389. *Other Arteries liable to,* 390. True Aneurism. *Formation and Proofs of its Reality,* 391. *Situation and size of,* 391. *Rupture into the Pericardium,* 392. *And Brain,* 392. *Coagula rare,* 393. False Aneurism, 393. *Formation,* 393. *Anatomy,* 394. *By Fissure,* 394. *Aneurism forming a new Arterial Channel,* 395. *Causes of Arterial Perforations,* 396. *Situations of False Aneurism,* 396. Mixed Aneurism. *Formation,* 397. *Anatomy,* 398. General Observations. *Aneurism by Hernia of the internal Membrane,* 398. *Solid Tumors formed by obliterated Aneurisms,* 399. *Parts involved in an Aneurismal Sac,* 399. *Its Size much determined by the Extensibility of these Parts,* 400. *Fibrinous Coagula in,* 400. *Aneurisms and Diseases of Arteries more frequent in Men,* 401.

ANEURISM ('Ανεύρυσμα, τος, τὸ, arteriæ dilatatio et inde ortus tumor, from 'Ανευρύνω̃, dilato, amplio) is an enlargement of a portion, or the whole, of the circumference of an artery.

Aneurisms of the aorta are divided by authors into four species.

1. *Dilatation*, which is an enlargement of the whole circumference of the artery.

2. *True aneurism*, which is a sacculated dilatation of a portion only of the circumference, or of one side of the artery.

3. *False aneurism*, which is formed by ulceration or rupture of the internal and middle coats, and expansion of the external or cellular into a sac. It is called *primitive* when all the coats are directly divided, as by a wound; and *consecutive* when it is consequent on ulceration or rupture of the internal and middle coats.

4. *Mixed aneurism*, which is a supervention of false upon true aneurism, or upon dilatation: that is, after dilatation either partial or general of all the three coats, the internal and middle burst, and the external alone expands into a further sac, surmounting the original dilatation or true aneurism.

1. *Dilatation, or enlargement of the whole circumference of the aorta.*

When the coats of the aorta, whether from inflammation or from any other morbid action, have become diseased, they lose their elasticity, a quality which resides principally in the middle tunic. As fluids press equally in every direction, the blood propelled by each contraction of the heart into the aorta, exerts not only a longitudinal, but a lateral force, which expands the vessel, and constantly tends to enlarge its caliber. The elasticity of the arterial walls enables the vessel to resist this expansive force, and to regain its previous caliber after the diastole. Consequently, when the elasticity is impaired or lost, the vessel, not being able to regain its original dimensions after each diastole, becomes permanently dilated, and this takes place to a greater or less extent, and with greater or less promptitude, in direct proportion to the predominance of the distending over the resisting force.

It very rarely happens that a dilated aorta does not present, in its interior, some of the morbid changes already described: (see ARTERITIS, p. 157.) namely, cartilaginous, steatomatous, atheromatous, or calcareous depositions, with a thickened, wrinkled, and fragile state of the internal coat. When such depositions are not apparent, the walls, according to my observation, are always more or less indurated, opake and inelastic; and also sometimes extenuated, particularly the middle coat, and sometimes thickened, with a softened and easily separable state of the internal coat: a condition of parts which is a much more natural cause of dilatation than paralysis of the middle coat, supposed by some authors to be its cause when no depositions are manifest.

The ascending portion and arch of the aorta, particularly the latter, are by far the most frequent seats of dilatation; but the descending portion, both in the chest and abdomen, is sometimes affected, and the dilatation is then either uniform throughout the whole length of the vessel, or it consists of one or even a series of ovoid, or fusiform expansions. The side of the artery adherent to the spine, and the lesser curvature of the arch, yield less readily than the other parts. Dilatation of the aorta does not in general exceed twice the natural caliber of the vessel, but I have occasionally seen it attain three, and even four times that size. When such is the case, it frequently presents many minor bulgings or pouches, which give it a considerable resemblance to the transverse arch of the colan. The walls of these pouches are often extenuated and semi-transparent from horn-like and calcareous depositions, and it is here more especially that mixed aneurism is apt to take place; for the brittleness of the depositions causes rupture of the internal and middle coats, and the engraftment of false aneurism upon the true.

Dilatation of the pulmonary artery is extremely rare. I have met with one remarkable case in which it was enlarged to four inches and a half in circumference. (Wetherall.)

Dilatations, even though pouched, scarcely ever contain laminated coagula; for the surface is in general too smooth to arrest the blood: when they do take place, it is in consequence of an ulcerated or fissured state of the internal membrane, which forms nuclei for the adhesion of fibrine.* The coagula thus formed occasionally fill up the whole of the dilated portion and leave the canal of the artery of its natural caliber.

The great arterial trunks rising at right angles from the aorta, as the innominata and cæliac, generally participate in the dilatation: the left subclavian almost always remains exempt; without doubt, says Laennec, on account of the acute angle at which it branches off.

Dilatation takes place not only in the aorta and its immediate trunks, but sometimes in smaller and more remote arteries, as, for example, the carotid by the side of the sella turcica, the temporal † and emulgent ‡ with their ramifications, the arteries of the extremities, and those feeding tumors of any description, particularly *fungus* hæmatodes and the hæmorrhagic nœvus or aneurism by anastamosis of John Bell.

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* Case by Burns. Diseases of the Heart, p. 206, and by Bertin and Bouillaud, Obs. xxxvi.

† *Cruveillier* Essai sur l'Anat. Patholog. Paris, 1816. tom. ii. p. 60.

‡ Journal de Méd. par MM. *Corvisart*, Leroux et Boyer, tom. vii. p. 255.

2. *True aneurism, or lateral, partial dilatation of the aorta.*

True aneurism differs from dilatation in the circumstances, that it is an enlargement of a limited portion only of the circumference of the aorta; that it generally rises with an abrupt margin; and that its neck is, in most cases, narrower than the body of the sac. (Case of Hill.) Its formation is to be attributed to a loss of elasticity and resistance in the particular part only that dilates; and the proofs of its existence consist in the possibility of tracing the internal and middle coats of the artery throughout the whole extent of the expansion, and in the presence within the sac of those morbid appearances, which are peculiar to the internal coats of arteries, such as calcareous, cartilaginous and atheromatous depositions, slight fissures and small red spots. These proofs have of late years been so frequently verified by dissection, that the reality of aneurism by dilatation of all the coats of an artery is no longer problematical.

Almost all the aneurisms of the ascending portion and arch are originally of the true species, but the false is sometimes engrafted upon them. The tumor generally springs from the anterior, or the lateral part of the vessel, while the posterior part is little, if at all implicated: it sometimes attains the magnitude of a mature fœtal head,* and generally inclines to the right side of the chest. When it springs from the root of the aorta, and the middle and internal coats happen to burst, there results, not a false aneurism surmounting the true, as in other parts, but a fatal extravasation into the pericardium. The reason of this is, that the part of the aorta referred to, is destitute of the cellular tunic, and the pericardium which supplies its place, not being equally extensible, bursts, rather than dilates into a false aneurism. In the same way, the deficiency of the cellular coat in the arteries of the brain, causes their rupture to be followed by an apoplectic extravasation, instead of by the formation of an aneurismal sac.

It has been stated by a recent writer that a preparation in Mr. Hunter's collection subverts the doctrine that "false aneurism does not form at the root of the aorta." The preparation of which he speaks, however, scarcely subverts this doctrine, since it is not one of *false* aneurism; for the middle coat is perfect, the internal one alone being either diseased, or removed, (which, is doubtful,) at the base of the sac. Though it has been denied by authors that *false* aneurism may form at the root of the aorta, it has not been denied that *true* may. I have myself seen it in more than one instance. Coagula are occasionally, but not often, found in true aneurisms; they are usually in masses, adherent by a peduncle, and seldom in layers investing the walls unless the aneurism is very large; the reason of which is, that, the mouth of the sac being in general spacious, the blood has a sufficiently free ingress and egress to circulate with force, while the surface of the sac is so smooth as not to arrest the fibrine and cause its deposition in layers. But when the circulation is by any cause enfeebled, the blood stagnates and forms coagula in masses, which become adherent by limited portions or peduncles. True aneurism is much more rare than either false, or dilatation.

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* *Corvisart*, Journ. de Méd. par MM. Corvisart. *Leroux* et *Boyer*, tom. vii. p. 355. *Laennec* de l'Auscult. tom. ii. p. 691.

3. *False aneurism, or aneurism by ulceration of the internal and middle coats.*

Nichols proved, by experiments made before the Royal Society of London, that when the internal and middle coats of an artery are divided, and water or air is forced into the vessel, the external coat is distended so as to form a small sac.* In the same manner, when the internal and middle coats are perforated by ulceration or a fissure, the blood, by its lateral pressure, gradually raises the external coat and expands it into a sac, which communicates by a narrow aperture or neck with the interior of the artery, whose caliber is not enlarged. As the distention proceeds, the external coat itself gives way, and the sheath of the vessel next opposes the effusion of blood: finally, when this also yields, the contiguous parts, whatever be their texture, contribute to the formation of the sac, they having previously undergone thickening and agglutination by chronic adhesive inflammation, to which distention had given rise.

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* Philosoph. Trans. vol. xxxv. p. 443.

Such is the manner in which the sac is formed in aneurism from ulceration of the arterial coats. It presents no vestige of the middle or fibrous coat, nor the depositions connected with the internal membrane: but its inner surface is extremely rugged and unequal from lymph irregularly deposited by inflammation. To this rugged surface adhere the layers of fibrine subsequently separated from the blood.

Perforation of the internal and middle coats is not always followed by aneurism of the kind described. Laennec met with a case in which the internal and middle coat had been divided by a narrow transverse fissure extending over two-thirds of the circumference of the artery, and the blood, instead of distending the external coat into a sac, had insinuated itself between it and the fibrous, and dissected them from each other over upwards of half the circumference of the artery, from the arch of the aorta to the common iliacs.* Fissures of the kind described result from cracks or lacerations following the direction of the fibres of the middle coat, or from wounds occasioned by calcareous depositions; but the case of Laennec, and two similar ones mentioned by Mr. Guthrie,† are the only instances within my knowledge in which a fissure has been followed by more than a circumscribed effusion of blood around it, occasioning a slight swelling of the external coat. Nichols found this in the body of George the Second,‡ and Hodgson once met with it.§

The late Mr. Shekelton has described, in the Dublin Hospital Reports, 3d volume, another, and previously unnoticed kind of aneurism: the blood had forced its way through the internal and middle coats, dissected the middle from the external or cellular for the space of four inches, and then burst again through the internal and middle coats into the canal of the artery, thus forming a new channel, which eventually superseded the old one, the latter having become obliterated by the pressure of the tumor.

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* De l'Auscult. tom. ii. p. 700.

† Guthrie, on the Diseases of Arteries, pp. 40 and 43.

‡ Philos. Trans. vol. lii. p. 269.

§ On Diseases of Arteries, p. 63.

The causes of perforation of the internal and middle coats and the formation of false aneurism, are, 1st. *ulceration*, generally occasioned by the detachment of calcareous incrustations, by atheromatous depositions under the internal membrane, and, more rarely, by tubercles, or small abscesses in the substance of the fibrous tunic: 2d. *rupture or cracking*, which takes place when the tunics have been deprived of their elasticity by cartilaginous, steatomatous, fungous and calcareous degeneration.* The immediate or exciting cause of the rupture, is generally some violent exertion or accident; and in most instances patients with aneurism date them from some occurrence of this kind. Rupture does not appear ever to take place in a perfectly sound artery; and if it did, the experiments of Dr. Jones prove that it would not be followed by an aneurism, as an effusion of lymph takes place, which strengthens the vessel in the lacerated part.†

While aneurisms of the ascending aorta and arch are, in the first instance, almost invariably true, though they occasionally become mixed; those of the descending aorta are generally false; and the caliber of the artery is, with few exceptions, not in the slightest degree dilated opposite to the tumor.

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* Scarpa, on Aneurism, § 20, 21, 22. Laennec de l'Auscult. tom. ii. p. 704. Hodgson, p. 62.

† Jones on Hæmorrhage, p. 125.

Aneurism by perforation of the internal and middle tunics, is the only species of which Scarpa admits the reality: but the inaccuracy of his opinions has been fully proved, and, as before stated, there is no longer any question respecting the actual existence of aneurism by dilatation of all the coats.

The cases of false aneurism that are on record, are very numerous. Reference may be made to the works of Lancisi, Morgagni, Guattoni, Scarpa, Desault, Warner, Hodgson, Horne, Laennec, Bertin and Bouillaud.

4. *Mixed aneurism, or false aneurism surmounting true.*

This species is formed in the following manner. All the three tunics of the artery first undergo an expansion which, according to its form, constitutes either a dilatation, or a true aneurism: as the expansion proceeds, the internal and middle tunics burst, and the external, being more extensible, dilates into a sac, surmounting the original enlargement.

Aneurisms of this description are very numerous. Whether the cyst succeeding a perforation of the arterial walls, has been preceded or not by their dilatation;—in other words, whether the aneurism is *true* or *mixed*, it communicates with the cavity of the aorta by an aperture more contracted than its body and circumscribed by a prominent border, corresponding with a kind of external strangulation. This disposition of parts has been perfectly described by Scarpa, and admirably represented in his plates.

General observations on aneurisms of the aorta.—Haller, and MM. Dubois and Dupuytren have remarked a variety of aneurism, in which the internal membrane makes a hernia through the ruptured fibrous coat and lines the sac, which is formed by the external or cellular coat. Hernia of the internal membrane may occur, according to Laennec, in very small aneurisms: he had seen it in two, which were not larger than cherries; but, when the tumor increases, the internal membrane speedily bursts. This he found to have been the case in two other aneurisms which did not exceed the size of walnuts.* The experiments of Mr. Hunter, Scarpa and Sir E. Horne prove, that when the external and middle coats of an artery are removed, the internal one does not dilate into an aneurism, but either bursts, or is strengthened by granulations arising from its surface, and by adhesions formed with the surrounding parts.

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* De l'Auscult, ii. p. 693.

Corvisart having found several firm, solid tumors of the size of nuts, intimately adherent to the aorta, while the external and middle coats appeared to be deficient at the point of attachment, was led to imagine that *extraneous* tumors, for such he conceived them to be, becoming adherent to arteries, led to the formation of aneurism.* Hodgson, on the contrary, regards the tumors in question as instances of aneurism cured, the sac having been filled up by lamellated coagula† and the volume of the tumor diminished by absorption; and Laennec, Bertin and the best authorities subscribe to his opinion.

As an aneurismal sac enlarges, the surrounding parts become involved in its composition. Thus, the bones, muscles and various other structures, often contribute to its formation. The viscera, also, become implicated when the tumor is situated in their vicinity; and the membranes with which they are invested being distended to their utmost, finally yield, and the sac bursts into their cavities. Accordingly, aneurisms frequently prove fatal by discharging their contents into the lungs, æsophagus, stomach, intestines, bladder, &c.

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* Essai sur les Maladies du Cœur, p. 313.

† On Diseases of Arteries, p. 127.

The size which the tumor attains depends upon the nature of the surrounding parts, and is very much determined by their extensibility—a property which is almost in direct proportion to the quantity of cellular tissue of which they are composed. Hence it is, that, when the disease is situated at the root of the aorta, where the pericardium supplies the place of the more extensible cellular coat of the vessel, the sac bursts into the pericardium before it has attained any great magnitude. Hence, also, it is, that, in the cranium, where the arteries are destitute of the cellular coat and are ill supported by the pia mater, and the soft pulpy substance of the brain, aneurism is extremely rare; for such a lesion of the coats of the arteries as would elsewhere give rise to aneurism, is here attended with rupture and apoplectic effusion. It has been already stated, however, that the arteries of the brain are not insusceptible of dilatation.

One of the first circumstances that almost invariably follows the formation of true aneurism, is, the deposition of the fibrine of the blood upon the internal surface of the sac. This deposition takes place in successive concentric layers, which have a different aspect according to the date of their formation. The most central consist simply of blood more or less firmly coagulated, and they are probably formed subsequent to death: a little farther, the coagulum is dryer, paler, and evidently composed of a large proportion of fibrine: still farther, are layers of pure, whitish, yellowish, or greyish fibrine; and finally, in contact with the walls of the cyst, are layers of the same matter, but completely opake, of a somewhat friable consistence like dryish paste, and very closely resembling flesh which has been deprived of its colour by boiling. The most recent layers adhere to each other so slightly as almost to float within the sac; those beneath are united by a downy or villous cellular tissue, the adhesion being stronger in proportion as the layers are older. Patches of vivid red, formed by reticulated blood vessels, are occasionally found in the fibrine, and blood often penetrates between its layers, and stains those which are friable, or decomposed. Coagula are softer in some cases than in others, though the physical circumstances be the same in both. The difference is probably owing to a difference in the chemical qualities of the blood.

From these anatomical characters, it is evident that lamellated coagula form by successive depositions of the fibrine of the blood; and the depositions are accounted for by the stagnation of the blood within the sac; for it is proved by experiment and observation that coagulation of this liquid takes place whenever its course is interrupted; hence the polypi that are found in the heart, the great veins, and the arteries, when the circulation through those parts is obstructed.

The coagulation of blood within a false aneurism is favoured by two circumstances—the narrowness of the aperture of communication with the artery, and the ruggedness of the interior of the sac. In true aneurism, as before stated, the width of the aperture of communication, and the smoothness of the interior of the sac, are unfavourable to the coagulation, and accordingly fibrinous *layers* are very seldom found in those aneurisms unless they be of great size, although they often contain coagula in masses attached at one part only by a peduncle of greater or less thickness.

The thickness of fibrinous depositions within aneurisms is sometimes very great. Most commonly it is from half an inch to an inch and a half, but I have seen it exceed three inches. The thickness is generally greater in one part of the sac than in another. Laennec has seen fibrinous coagula as compact and diaphanous as horn softened to the utmost by heat, and of a thickness exceeding five fingers' breadth.

Aneurisms, and the diseases of the coats of arteries which precede their formation, are much more frequent in men than in women. Of sixty-three cases seen by Hodgson, fifty-six occurred in the former and only seven in the latter.* I have found the proportion in females rather larger than this, with respect to aneurism of the aorta; but with respect to external aneurism it is much smaller, perhaps not exceeding one in fifteen to twenty.

SECTION II.

PATHOLOGICAL EFFECTS OF ANEURISMS OF THE AORTA ON CONTIGUOUS PARTS.

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* On Diseases of Arteries, p. 87.

Bones are most exposed to Erosion, 411. *By Aneurisms in what Situations*, 411. *When the Periosteum contributes to the Sac, it secretes Bone*, 412. *Small Aneurisms erode Bones more than large, why*, 413.

The pathological effects of aneurisms of the aorta on contiguous parts vary according to the volume, the form, and the position of the tumor.

Dilatation, when not very considerable, produces little derangement of the surrounding parts. For, as the swelling is equable and diffuse, it does not exert a pressure on any one organ in particular, and its magnitude is not such as to create much inconvenience from general infarction. The worst of its effects are those which it produces on the trachea and great bronchi; for though the pressure be slight, it often suffices, in consequence of the great irritability of these parts to occasion considerable dyspnœa. It must not, however, be imagined that dilatation is an unimportant affection. It will hereafter be shown that when complicated with enlargement of the heart, which it generally brings on, it is one of the most formidable diseases incident to the circulatory apparatus.

An aneurism which forms a defined tumor, whether it be of the true, or the false species; whether it be large, or small, may produce the most pernicious effects. These are,

1st. Such as result from compression of the neighbouring parts.

2nd. Such as result from their destruction.

1st. By compression, the functions of the lungs, bronchi, heart and œsophagus, are deranged, and that sometimes to a fatal extent. In the abdomen the functional derangements are comparatively inconsiderable, and very rarely endanger life. The reason of this is two-fold; first, that the abdominal organs are not of so vital a nature as the thoracic; and secondly, that the tumor, instead of being pent up in a rigid, bony case like the chest, is permitted by the yielding of the intestines, and the distensibility of the abdominal parietes, to expand freely in almost every direction. Pressure on any particular organ, therefore, is in a great measure obviated by the want of counter-pressure or a fulcrum. Ventral aneurism, however, sometimes deranges the respiration by preventing the due descent of the diaphragm—an effect which may proceed either from the magnitude alone of the tumor, or, what is much more common, from its being seated near, or in the substance of the muscle, and impeding its motions. Ventral aneurism is also occasionally characterized by involuntary evacuation of the urine and fœces, by remarkable alternations of constipation and diarrhœa, and by deep-seated excruciating pains, resembling those of humbar abscess. These symptoms arise from compression of the nerves, particularly the hypogastric plexus around the aorta.

2nd. The consequences of destruction of contiguous parts are far more formidable than those resulting from their compression.

When the tumor exerts an unusual pressure on any organ or texture, adhesive inflammation takes place and unites the parts in contact. As the pressure increases, absorption and ultimately perforation of the sac ensues, and death from internal hæmorrhage is the immediate consequence. The perforation takes place either by sloughing or by laceration, according to the nature of the membrane or texture perforated. Thus, when the tumor advances to the skin or when it extends into a cavity lined by a *mucous* membrane, it bursts by the separation of a slough which has formed upon its most distended parts, and not by laceration. On the contrary, when the sac projects into a cavity lined by a *serous* membrane, sloughing of the membrane does not take place, but the parietes of the tumor, having become extremely thin in consequence of distention, at length burst by a crack or fissure, through which the blood is discharged. An aneurism may burst into a great variety of parts, which we shall notice in succession.

When the lungs are in contact with the tumor, adhesion, absorption of the sac, and rupture of the pleura take place, and the effused blood deluges the bronchi and causes suffocation. (Lafin.)

It often happens that an aneurism of the ascending aorta, or arch, compressing the trachea or one of the great bronchial trunks, opens its way into it by ulceration of the cartilaginous rings and sloughing of the mucous membrane, and causes suddenly fatal hæmoptysis.

More rarely, perforation takes place into the œsophagus, and death then ensues from hæmetemesis.

Aneurisms occasionally burst at the origin of the aorta, and cause death by effusion of blood into the pericardium. The fatal event, however, is not always so sudden as in the preceding cases; a circumstance which Laennec attributes to the pericardium being supported, and the effusion consequently restrained, by the general infarction of the chest resulting from the presence of the tumor. This reason appears to me unsatisfactory, because, as before explained, aneurisms at the root of the aorta generally burst before they attain any considerable magnitude: nor, if large, would the resistance offered by the atmospheric pressure in the lungs equal the force with which the blood tends to escape into the pericardium—a force equal to the propulsive power of the left ventricle. It is, perhaps, more probable that the inextensibility of the pericardium beyond a certain point, and the resistance of the heart to compression, form the principal powers which limit the effusion of blood.

It would appear that life is sometimes protracted for a considerable period after the rupture of the sac; for in specimens presented to the Société de la Faculté de Médicine by M. Marjolin, the margins of the aperture, according to Laennec, were polished, as if of old standing and, as it were, fistulous.[*]

Rupture into the pericardium is very rare. Laennec never met with an instance. The first that has fallen under my own observation, occurred recently at St. George's Hospital. Morgagni[†] and Scarpa,[‡] however, have collected together a considerable number of these cases, and Hodgson saw two, the aneurism beginning half an inch above the semilunar valves and occupying the whole ascending aorta and arch.

Aneurisms have been known, though very rarely, to burst into the pulmonary artery. MM. Payen and Zeink saw an instance,[§] and Dr. Wills another.[||] My friend, Professor Monro, showed me a preparation of an aneurismal pouch springing from the aorta directly against the pulmonary artery;

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[*] Laennec Op. Cit. ii. p. 715.

[†] Epist. xxvi. Nos. 7, 17, 21.

[‡] On Aneurism, § xix. p. 103 et sequent.

[§] Bulletin de la Faculté de Médicine, 1819, No. 3.

[||] Trans. of a Soc. for the improvement of Med. Chirurg. Knowledge, vol. iii. p. 85.

and it is probable that, if the patient's life had been prolonged, rupture would have taken place into the artery.

The left cavity of the pleura and the posterior mediastinum are the parts into which thoracic aneurisms most frequently burst. It is extremely seldom, on the contrary, that they open into the right pleura.

Laennec has seen an aneurism of the descending aorta, which had compressed and destroyed the thoracic duct and produced engorgement of all the lacteal vessels.

Aneurisms sometimes compress the descending vena cava and cause cerebral congestion, œdematous intumescence of the face and even apoplexy. I have met with several instances of this kind. Corvisart,* and Bertin and Bouillaud † each cite a case of apoplexy thus occasioned.

Another effect of aneurisms is, to obliterate arteries springing from them or contiguous to them. I have met with two cases in which both the left carotid and subclavian were plugged up at their origin from the tumor.‡ The obliteration is sometimes effected, not by a plug of lymph, but by contortion or compression of the vessel. Mere contraction of the origins of arteries from these causes is very common.

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* Journal de Médicine, par MM. Corvisart, Leroux et Boyer, tom. xii. p. 159.

† Traité des Maladies du Cœur, p. 137.

‡ Case of Aneurism, vii. viii. Lond. Med. Gaz. Sept. 12, 1829, p. 449.

Ventral aneurisms may open their way into the various abdominal viscera, as the intestines, the bladder, &c.

Aneurisms not only cause destruction of the soft parts; but, what is still more remarkable, erosion of the bones. This phenomenon has been variously explained. The old pathologists erroneously ascribed it to a chemical solvent power of the blood. Hunter, Scarpa and Hodgson thought that it resulted from absorption of the earthy matter, in consequence of the pressure of the sac. Corvisart and Laennec attribute it to a sort of detrition or wearing down produced by a purely mechanical action. Bertin and Bouillaud believe that it is more or less dependent on inflammation. To myself it appears that absorption and mechanical detrition are the principal agents concerned in producing the effect. That pressure is capable of exciting absorption of bone, is certain, as the vertebræ have been found excavated by an aneurismal tumor without being divested of their periosteum;* and there can scarcely be a doubt that, when a denuded bone is exposed to the constant dashing of a column of blood, it undergoes disintegration by the mechanical detachment of its particles.

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* Hodgson, p. 79.

Whether inflammation ever contributes to the effect, is difficult positively to determine. Appearances, however, are adverse to this opinion, as pus has never been found on bone eroded by an aneurism; as exfoliation scarcely ever takes place, and as nothing is discovered on it analogous to the cicatrization or irregular reproduction observable in other bones when affected with caries.

Cartilage, whether exposed to the action of the blood in aneurismal sacs, or to the pressure alone of the tumor, either remains entirely uninjured, or suffers incomparably less than bone. This is most manifest in the intervertebral substance and the cartilages of the false ribs. The circumstance is attributable to the elasticity of cartilage, which protects it from mechanical disintegration, and to its almost inorganic structure, which renders it little susceptible of absorption, or ulceration. The bones liable from their position to be eroded by aneurism, are, the vertebræ, the sternum, the ribs, and sometimes the ossa ilii.

It is principally by aneurisms of the descending aorta that the vertebræ are injured. In these cases the portion of the sac in contact with the vertebræ is entirely destroyed, and its borders adhere very firmly around the eroded part of the bone, on which the blood plays freely in consequence of the fibrinous layers having been absorbed at that part. The destruction is sometimes so deep that the shell of the vertebræ forms the only partition between the sac, and the spinal canal. Very rarely, however, does rupture take place into the canal. I am not aware that there are more than two instances on record; one by Laennec, in the Révue Médical for 1825; and another of which the preparation, by Mr. Chandler, is in the Hunterian Museum.

Ventral aneurisms seldom produce erosion of the bones; because the abdominal viscera and walls yield to the tumor.

It is by aneurisms of the ascending aorta that the sternum and ribs are eroded, and the tumor generally presents on the right side. Aneurisms of the arch and the innominata, project at the upper part of the sternum, and about the clavicles, which they have been known to dislocate at their sternal extremities. When the tumor is connected with the posterior part of the arch, it shews itself underneath the left clavicle.

According to Hodgson, when the periosteum contributes to the formation of the sac, its vessels continue to secrete an earthy matter, which, in some instances, has been deposited to such an extent as to form a considerable portion of the tumor.

Small aneurisms have the effect of destroying bones in a greater degree than large: a circumstance attributable to the greater concentration of the pressure exercised by them.

SECTION III.

SIGNS AND DIAGNOSIS OF ANEURISM OF THE AORTA.

In the present section, the general and physical signs will be described separately: in the next, a brief synopsis will be given of the two conjointly, with reference to the several forms of aneurism.

General Signs. *Impenetrably obscure*, 414. *Only one that is certain*, 414. *General Signs of Disease of the Heart not pathognomonic of Aneurism*, 415. *The more characteristic Signs ambiguous unless corroborated by the physical*, 415. *The more characteristic Signs with their Fallacies described*, 416: *viz.* 1*st*. *Infarction of the Chest*, 416; 2*nd*. *Dissimilarity of the Pulses*, 416; 3*rd*. *Pulse later than the ventricular Systole*, 416; 4*th*. *Purring Tremor of the Sternum*, 417; 5*th*. *Wheezing Respiration with croaking or whispering Voice*, 419; 6*th*. *Terebrating Pain in the Spine and aching of the Shoulder and Arm*, 420; 7*th*. *Feeling of Ebullition in the Lungs*, 420; 8*th*. *Spasm in the Course of the Diaphragm*, 421; 9*th*. *Pulsation under the Sternum*, 421; 10*th*. *Pulsation above the Sternum* *and Clavicles*, 422; 11*th. Dulness of the Chest on Percussion*, 423. PHYSICAL SIGNS. *Researches of Laennec inconclusive*, 423. *Summary of the Signs discovered by him*, 424. *Signs by the Writer*, 425; 1*st. The aneurismal Sound*, 425; 2*nd. Its Situation*, 425; 3*rd. The second Sound*, 426; 4*th. Peculiarities of the aneurismal Sound, and their Causes*, 426. *From what Causes the Sound is the loudest*, 428. *Sound in old thick Aneurisms*, 428. *Sound on the Back*, 429. 5*th. Purring Tremor*, 429; *Its Cause*, 429; *Its Situation*, 430. *Aneurismal Pulsation*, 431; *Where felt*, 431.

1. *General Signs of Aneurism of the Aorta.*

WHEN an aneurism is buried deep in the chest, and not capable of being detected by the sight and touch, it does not present a single general sign which is peculiar to itself and therefore pathognomonic of its existence. There are even cases in which it occasions no functional derangement—no inconvenience whatever; and the first circumstance that unveils the truth is, the sudden death of the patient while apparently in the enjoyment of perfect health. I have met with six or seven instances in which large aneurisms have existed without awakening even a suspicion in the minds of the medical attendant. One, in particular, eluded the penetration of a distinguished foreign auscultator, though he explored the lungs with eminent success.

There is only one general sign of aneurism of the thoracic aorta which is unequivocal and certain:

namely, a tumor presenting externally and offering an expansive as well as heaving pulsation, synchronous with the action of the heart.

Of the remaining general signs, a large class are identical with those of organic disease of the heart: viz. palpitation, dyspnœa, cough, tendency to syncope, terrific dreams, starting from sleep, hæmoptysis, livid or otherwise discoloured complexion, cerebral or hepatic congestions, serous infiltration, &c. This identity arises from an identity of cause; namely, an obstacle to the circulation, which depends either upon the aneurism alone, or conjointly upon it and a disease of the heart, to which, sooner or later, the aneurism almost invariably gives birth. It is obvious, therefore, that the signs of this class are equivocal.

There are, however, certain other general signs which are more characteristic: yet even these are ambiguous and unsatisfactory; as they only bespeak lesions of the viscera, or derangement of their functions, but do not proclaim the latent cause of the mischief. But when they coincide with the signs derived from auscultation they lose their ambiguity and rise into real importance; for the two classes of signs, general and stethoscopic, are a commentary on each other, and reciprocally borrow a precision and certainty of which they are individually destitute.

I shall succinctly describe the general signs to which I refer, and subjoin to each the principal sources of fallacy. The means of detecting the latter, I shall point out in the final synopsis.

1. When the tumor has attained a considerable magnitude, the cavity of the chest is preternaturally crowded, and the patient complains of a sense of constriction, infarction, and oppression.

But these sensations are common to almost all diseases of the chest.

2. The radial pulses are sometimes dissimilar, or one is extinct—an effect dependent on obstruction, or obliteration, of the arteria innominata, or left subclavian.

But the difference of the two pulses at the wrist may proceed from a variety of causes independent of aneurism of the aorta, as, contraction of the origin of either subclavian from osseous, cartilaginous, steatomatous, or other depositions; obstructions in the course of the artery, occasioned by tumors, wounds, subclavian aneurism, &c.; an irregular subdivision of the humeral, brachial, or radial artery. I have known the most ludicrous surmises occasioned by the radial crossing to the outside at the middle of the fore-arm, and the superficialis volæ supplying its place at the wrist.

3. When the origin of either subclavian is con-

tracted, the pulse at the corresponding wrist is a little later than the ventricular systole.

I have not found this symptom uniformly present. The heart is more frequently its source than the aorta, and I have observed it to be most considerable in cases of regurgitation into the left auricle; but obstruction of the aortic valves may occasion it in a minor degree, particularly if this lesion be accompanied with attenuation or atony of the ventricular parietes. When the sign exists in both pulses, the presumption is strong that its source is in the heart.

4. According to Corvisart, a purring tremor—the *frémissement cataire* of Laennec—is sometimes perceptible to the hand at the middle or upper part of the sternum, and indicates aneurism of the ascending aorta.

Purring tremor above the clavicles, is an almost constant concomitant, and therefore a valuable sign, of dilatation of the arch; but, according to my experience, it is unfrequently and imperfectly occasioned by sacculated aneurisms, especially if lined by strata of lymph. I have never known the tremor to be occasioned below the clavicles by dilatation, unless the enlargement was so great as to extend beyond the lateral margins of the sternum, and allow the tremor to be felt through the intercostal spaces: but I have met with one case in which a dilatation of the pulmonary artery, though not voluminous, afforded a marked tremor between the cartilages of the second and third ribs on the left side: this, however, is not remarkable, as the artery *naturally* lies nearly opposite to the part described. I have never known a sacculated aneurism create a tremor below the clavicles, unless the tumor had eroded the bones of the chest and presented externally underneath the integuments.

But the purring tremor may be occasioned in any part of the chest by mucous rattles, particularly those of the snoring kind, in the large bronchial tubes; and I have observed that, when derived from this source, it is a very common cause of deception, in reference both to aneurisms of the aorta, and ossifications of the heart. Purring tremor of the pulse is regarded as a sign, though it is a fallacious one, of ossification of the aortic valves. From many dissections it has appeared to me to be generally connected with two circumstances; viz. a powerful action of the heart, and ruggedness, without appreciable contraction, of the aortic orifice, or interior of the vessel. It, therefore, seldom exists unless either the action of the heart be accelerated, or the left ventricle hypertrophous. It may also be produced independent of organic disease, by mere velocity of the circulation. (See p. 72.)

5. When the trachea, or primary bronchial divisions are compressed by an aneurismal tumor, a harsh wheezing or sibilous sound, proceeding deep from the throat, characterizes the respiration; the voice is either croaking, or reduced to a whisper, or it is a compound of both; the breathing is often extremely laborious, and when the heart is simultaneously diseased, dyspnœa sometimes occurs in paroxysms of the most suffocating severity. When the œsophagus is compressed, deglutition of solids is rendered difficult, and sometimes impracticable; for the descent of the morsel excites an excruciating pain from the summit of the sternum to the spine, or lancinating deeply in every direction through the chest.

But compression of the trachea, or œsophagus, with the above symptoms, may be occasioned by tumors of any description. Wheezing respiration may proceed from an accumulation of glutinous mucus in the great bronchi. I have likewise known it produced in an extreme degree by laryngitis with thickening of the soft parts covering the arytænoid cartilages, and also by ossification and ulceration of the larynx from stumous, syphilitic, and mercurial disease. So difficult is it to distinguish the seat of wheezing respiration, that it has in many instances been imputed to an affection of the larynx when it was in reality occasioned by an aneurism of the aorta, and bronchotomy has several times been actually performed with the view of obviating suffocation.

6. When the vertebræ are eroded, the patient suffers an intense terebrating pain in the spine; and when the brachial plexus of nerves is compressed by the tumor, an aching sensation pervades the left shoulder, neck, scapula and arm, with numbness, formication, and impaired motive power of the limb.

But I have met with cases in which nearly similar pains were experienced, although there was no destruction of the vertebræ; and it is common to hear individuals affected with rheumatism or spinal disease make the same complaints. The affection of the arm may be occasioned by various forms of organic disease of the heart, and it thus constitutes a part of that concatenation of symptoms which is denominated angina pectoris. I have likewise often met with it in hysterical females subject to palpitation and occasionally in cases of pericarditis. In all these cases the pain probably originates in irritation of the cardiac plexus of the sympathetic propagated to the brachial plexus.

7. When in consequence of an adhesion between the aneurismal sac and the pleura, the blood plays upon the lungs, a sense of ebullition is said to be experienced.

But the same symptom is familiar to individuals labouring under phthisis, or chronic mucous catarrh; and it proceeds from the successive bursting of large bubbles, formed by the transmission of air through the fluid in tuberculous caverns, or in the greater bronchial ramifications.

8. It occasionally happens that the patient suffers excruciating pain from a spasm, pursuing the course of the diaphragm, and binding the chest around, as with a cord.

This symptom is too vague to be important, and it also occurs in hysteria, gastrodynia, colic, spinal diseases, and rheumatism of the diaphragm.

9. A pulsation felt underneath the sternum or ribs at the superior part of the chest.

This, although one of the least equivocal signs of aneurism, is not without ambiguity. It may be occasioned by a tumor of any description, as an enlarged gland, or a cancer, interposed between the sternum and the aorta, and receiving the pulsation of the latter. Even Dr. Baillie says "but we are not to conclude from this symptom (viz. pulsation at the superior part of the chest) that there is certainly an aneurism. I have felt the same kind of pulsation in other cases; as, for instance, where the pericardium was found strongly to adhere to the heart, where there was a slight inflammation upon the surface of the heart, with a little more water than usual in the pericardium; and where a morbid enlargement had taken place in the heart, without any aneurismal swelling." Every one much conversant with disease must have made the same observations.

10. A pulsation is felt above the sternum or clavicles.

But this may be occasioned, 1st, by enlarged glands or other tumors seated on the subclavian artery, and receiving its pulsation; 2d, by varix of the jugular vein at its junction with the subclavian; both of which conditions have deceived expert practitioners; 3d, by subclavian aneurism. This affection sometimes resembles aneurism of the aorta so exactly, that it is extremely difficult to distinguish them. Allen Burns records a case in which all the eminent surgeons of the district were unanimous in pronouncing the affection subclavian aneurism; yet it proved to be aortic.* Sir A. Cooper has published a number of similar cases; and one is mentioned by Professor Monro tertius.† 4th, A pulsation above the sternum or clavicles may be occasioned by carotid aneurism. This also may readily be confounded with aneurism of the aorta, or of the subclavian artery. In

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* Surg. Anat. of head and neck, p. 30.

† Elements of Anat. vol. ii. p. 249.

April, 1826, I saw a case at Guy's Hospital, which led to much deliberation respecting the propriety of taking up the carotid above a pulsating tumor, supposed to be an aneurism of that artery. It was finally decided that the tumor was too low, and the design was judiciously abandoned. The affection proved to be a dilatation of the aorta, and arteria innominata. The carotid was sound. This state of parts was indicated to me by the stethoscope. Mr. Hodgson met with a similar case.*

11. The superior and middle parts of the chest are dull on percussion. But this sign is common to an infinity of other diseases, and the resonance is seldom impaired unless the aneurism be very large.

It cannot be a subject of surprise, that a series of symptoms, liable to so many fallacies should have proved insufficient, without the aid of auscultation, to dissipate the deep obscurity which involved the diagnosis of aneurisms of the aorta.

Physical signs of aneurism of the aorta.—The investigations of M. Laennec on aneurism of the thoracic aorta, were limited and inconclusive. Accordingly, he remarks that, " Of all the severe lesions of the thoracic organs, three alone remain without pathognomonic signs to a practitioner expert in auscultation and percussion—namely, aneurism of the aorta, pericarditis, and concretions of blood in the heart previous to death.

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* On the Diseases of Arteries, p. 90.

I shall first present the opinions of Laennec, respecting the physical signs of aneurism of the aorta, and then offer the results of my own researches, by which I hope to make it apparent that this malady is characterized by sufficiently pathognomonic signs.

Laennec's opinions are as follows,—On applying the cylinder, in two instances, to tumors presenting externally, he found that their pulsations were exactly isochronous with the pulse; that the shock and sound greatly exceeded those of the ventricles; that the beating was distinctly audible on the back, and that the second sound could not be distinguished at all. For the last reason he denominated the aneurismal pulsation *simple*, in contra-distinction to that of the heart, which has a double sound, in consequence of the alternate systole and diastole of the ventricles. From these two cases he felt certain that, in some instances, pectoral aneurisms might be recognized by the *simple pulsation* usually much stronger than that of the heart; but he thought that in a larger proportion of cases, the sign would be insufficient: for, as the slightest dilatation of the heart renders its sounds audible over the whole sternum and even below and along the clavicles, he imagined that, under such circumstances, the first or systolic sound of the organ would be confounded with the sound of the aneurism, with which it is synchronous; while the second or diastolic sound, being audible as far as the tumor, would lead the auscultator to suppose that he there heard the beating of the heart, and not that of the aneurism. I shall presently shew that this reasoning is incorrect.

As the second sound is not audible over the abdomen, Laennec found no difficulty in recognizing ventral aneurisms by the *simple pulsation*.

According to my experience, the cylinder is scarcely less capable of affording decisive indications of pectoral, than of ventral aneurism. It is unimportant whether the pulsations be "*simple*" or "*double*," for, though double, they may be distinguished from the beating of the heart, by unequivocal criteria: viz.

1st. The first aneurismal sound, coinciding with the pulse, is invariably louder than the healthy ventricular sound, and generally than the most considerable bellows-murmurs of the ventricles.

2nd. On exploring the aneurismal sound from its source towards the region of the heart, it is found to decrease progressively, until it either becomes totally inaudible, or is lost in the predominance of the ventricular sound. Now, if the sound emanated from the heart alone, instead of decreasing, it would increase on approximating towards the præcordial region.

3d. The second sound actually does sustain this progressive augmentation on advancing towards the heart; and as its nature and rhythm are found to be precisely similar to those of the ventricular diastole heard in the præcordial region, it is distinctly identified as the diastolic sound. The second sound, therefore, corroborates, rather than invalidates the evidence of aneurism afforded by the first; for, if both sounds proceeded from the heart, both would, on approximating towards it or receding from, it, sustain the same progressive changes of intensity.

4th. Another distinctive characteristic of the aneurismal pulsation is, the peculiar nature of its sound. It is a deep, hoarse tone, of short duration, with an abrupt commencement and termination, and generally louder than the most considerable bellows-murmurs of the heart. It accurately resembles the rasping of a sounding-board heard from a distance; whereas, the sound occasioned by valvular disease of the heart has more analogy to the bellows-murmur, being somewhat soft and prolonged, with a gradual swell and fall.

It appears probable that the greater hoarseness and loudness of the aneurismal sound above, than below the clavicles, is attributable to its being reverberated through the chest before it arrives at the ear. This probability is countenanced by the following considerations: a. That, in several cases with which I have met,* while the sound, above the right clavicle, was loud and hoarse, it was merely a whizzing without hoarseness, on the superior part of the sternum, where the dilated ascending aorta was in apposition with the bone, and where, consequently, the sound was transmitted immediately to the ear. b. That, in the heart, the proximity of which organ to the thoracic parietes is unfavorable to the expansion and reverberation of its sounds, morbid murmurs are less hoarse and loud than those occasioned by pectoral aneurisms. That, in aneurisms of the abdomen and extremities, where there is little or no reverberation of sounds, there is a still less degree of hoarseness and loudness.

The abruptness of the aneurismal sound, compared with the prolonged, swelling character of the ventricular murmur, is owing to the latter being generated by a gradual muscular contraction, while the former is due to the sudden propulsion of a fluid through a tube naturally very resistant, and rendered still more unyielding by disease.

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* See, for instance, Case 9, *Lond. Med. Gaz.* Sept. 12, 1829.

The loudest aneurismal sound is that occasioned by dilatation: and it has more of the grating or rasping character, in proportion as the interior of the vessel is more overspread with hard, and especially osseous asperities. When the dilatation is confined to the ascending aorta, the sound, impulse, and purring tremor are stronger on the right, than on the left side of the neck; and the sound along the mesial part of the sternum—the tract of the ascending aorta—is superficial, and of a whizzing or hissing character.

Old aneurisms, the parietes of which are thickened by fibrinous depositions, yield only a dull and remote sound. In all cases of dilatation, and in the majority of sacculated aneurisms, the sound is loudest above the clavicles, even though the impulse be stronger below. In some cases of the sacculated species, it is louder on the side of the neck opposite to that where the tumor exists. I have found this to proceed from one or other of two causes—first, disease of the inner coat of the aorta before or beyond the tumor and opposite to that side of the neck where the sound was loudest; secondly, the interposition of the sac, thickened with fibrinous layers, between the aorta and the superclavicular region, in consequence of which the source of sound,—the mouth and cavity of the sac, was unusually remote on the side occupied by the tumor.

The sound of aneurisms is in most instances audible on the back; and when the tumor occupies the descending aorta, and is extended along the spine, it is often louder behind than on the breast. If it possesses on the back the abrupt, rasping character, the evidence which it affords is almost positive; for the loudest sounds of the heart, when heard on the back, are so softened and subdued by distance, as totally to lose their harshness.

5th. *Purring tremor* is another characteristic of the aneurismal pulsation. It is more considerable in simple dilatation than in sacculated aneurism, particularly if the former be accompanied with much asperity of the internal membrane. From numerous dissections, the fact appears to me to admit of the following explanation:—in cases of dilatation the interior of the vessel is almost invariably rendered rugged by osseous, cartilaginous, or other adventitious depositions; and the blood in permeating such a tube necessarily occasions a strong tremor, as its particles are thrown into preternatural commotion and collision, not only by the enlargement of the caliber of the vessel at the dilated part, by which they are diverted from their direct course, but also by the roughness of the surface of the vessel, by which they are reflected in counter-currents from its sides. In sacculated aneurism, on the contrary, though a portion of blood descends into the sac, the greater quantity pursues a direct and tranquil course through the smooth canal of the artery, and the tremor is therefore less considerable.

I have uniformly found the purring tremor confined to the supra-clavicular regions, except in the case of aneurisms which had protruded through the ribs and presented immediately underneath the integuments. It is rarely occasioned at all by old aneurisms; because, in consequence of their magnitude and the thickening of their sacs with fibrinous coagula, they possess little susceptibility of vibration.

Purring tremor proceeding from organic disease of the aorta, may easily be distinguished from that occasioned by nervous agitation. The former is constant, or may be excited at pleasure simply by accelerating the circulation; it is restricted to a limited space above the sternal extremities of the clavicles, and is accompanied with the hoarse aneurismal sound. Nervous purring tremor, on the other hand, is only occasional, occurring when there is an exacerbation of nervous excitement and restlessness; it extensively pervades the adjoining arteries, and the concomitant sound is comparatively soft and feeble.

Pulsation attends every species of enlargement of the aorta. In dilatation, it exists only above the sternal ends of the clavicles, and always on both sides of the neck simultaneously; though, when the enlargement is confined to the ascending aorta, it is stronger on the right, than on the left side. When dilatation is of a pouched form, and of great magnitude, it may occasion pulsation under the sternum. Of this I have met with instances. Carotid and subclavian aneurisms produce impulse, sound, and tremor on the affected side only, and by this circumstance they may easily be discriminated from aortic enlargements.

In sacculated aneurism seated in the upper parts of the chest, pulsation exists both above and below the clavicles, but I have generally found it stronger below. When the tremor is large, and occupies the left extremity of the arch, the impulse is often perceptible from the sternum to the left shoulder, and as low down as the third or fourth rib. When it lies in contact with the ribs posteriorly, the shock is sometimes felt on the back. This, however, is a rare occurrence. A pulsation under the sternum or ribs is one of the least ambiguous signs of sacculated aneurism.

SECTION IV.

SYNOPSIS OF THE PHYSICAL, IN CONJUNCTION WITH THE GENERAL SIGNS, IN REFERENCE TO THE SEVERAL VARIETIES OF ANEURISMS OF THE AORTA.

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Nervous Pulsation of the Abdominal Aorta. *Diagnosis from Aneurism by* Physical Signs, 443; *by* General Signs, 443.

Simple dilatation of the arch, and ascending aorta.

Physical signs.—1. A constant pulsation above both clavicles at their sternal ends; stronger on the right side if the enlargement is confined to the ascending portion, and never communicated to the sternum or ribs, unless the dilatation be enormous.

2. A hoarse rasping sound above both clavicles, of brief duration, commencing and terminating abruptly. If the enlargement is confined to the ascending portion, the sound is louder above the right, than above the left clavicle, and, along the middle of the sternum, it is superficial and of a hissing or whizzing character; by which, and by the sound being situated higher up the chest, it is distinguishable from that of valvular disease. It is usually distinct on the back, where the ventricular sounds, if audible at all, are very obscure.

3. A purring tremor above the clavicles but never below. It is stronger, and the concomitant sound more grating, in proportion as the interior of the aorta is more overspread with hard, and especially osseous inequalities.

General signs of dilatation.—Frequently none. When any exist, they are a slight degree of those common to all organic diseases of the heart, viz. the signs of an embarrassed circulation. They assume a most aggravated aspect when dilatation becomes complicated with organic disease of the heart.

Fallacies, and methods of detecting them.

a. *Nervous arterial excitement and reaction after loss of blood*, sometimes occasion an impulse and bellows-sound above the clavicles; but they may be discriminated by the impulse being feebler and the sound more hissing or whizzing than in aneurism of the aorta, and by the absence of purring tremor. It is, in fact, in the subclavian arteries that the phenomena take place; for though the aorta be under the same excitement, its action is not so violent as to extend in any appreciable degree to the supra-clavicular regions.

b. *Adhesion of the pericardium*, particularly when accompanied with much hypertrophy of the heart, I have in many instances found to occasion the impulse and whizzing sound above the clavicles in a still more remarkable degree than nervous excitement. The phenomena depend upon the suddenness and, as it were, spasmodic energy of the ventricular contraction—a subject already considered. (p. 131.) They may be distinguished by the sound being more whizzing and less hoarse, and the impulse more jerking, than in dilatation of the aorta; and they should always be suspected to proceed from adhesion when the heart presents the signs, and the history affords the presumption of that affection.

c. *Dilatation of the pulmonary artery* is a third, though extremely infrequent source of fallacy; for the mode of detecting it I refer the reader to the next head: viz.

Dilatation of the pulmonary artery.

Physical signs.—I have met with one case in which this artery was dilated to the extent of five inches in its internal circumference. It presented the following physical signs, which have not hitherto, I believe, been noticed.

1. A pulsation with purring tremor between the cartilages of the second and third ribs on the left side, and thence in a decreasing degree downwards, but not appreciable above the clavicles. Also a slight prominence between the same ribs.
2. An extremely loud, superficial, harsh, sawing sound, audible above the clavicles and over the whole præcordial region, but loudest on the prominence between the second and third ribs.

The general signs were those of hypertrophy and dilatation of the heart, with which the dilatation of the pulmonary artery was complicated.

Fallacies.—Dilatation and aneurism of the aorta are perhaps the only affections for which dilatation of the pulmonary artery could be mistaken. The signs, however, of the latter are so characteristic that, with due attention, I think it scarcely possible to commit an error. Namely, a pulsation between the cartilages of the second and third ribs could not be occasioned by a dilatation of the ascending aorta; as this artery, even when dilated, is too far to the right to extend beyond the margin of the sternum. Again, a sacculated aneurism of the ascending aorta could not reach the cartilages of the second and third left ribs without being very large and in this case it would form a much greater tumor externally than existed in the present instance. The sound also of such an aneurism would be dull and as if remote, instead of loud and superficial. Finally, either a dilatation or an aneurism of the aorta would occasion a greater pulsation and sound above one or both clavicles, than existed in the case of which we speak.

Sacculated Aneurism of the thoracic Aorta.

Physical signs.—1. A pulsation both above and below the clavicles, but usually stronger below. If the tumor occupies the ascending aorta, its impulse is most perceptible on the sternum and towards its right. If it is seated in the arch or commencement of the descent the pulsation inclines to the left side, and sometimes reaches to the shoulder. It is occasionally perceptible on the back. In front the pulsation is always stronger on the tumor, than at some point intermediate between it and the heart, and generally stronger than the impulse of the heart itself.

2. The aneurismal sounds described under dilatation, but weaker. In large, old aneurisms it has a dull and remote character, and is sometimes louder on the side of the neck opposite to that where the tumor is situated. It is generally audible on the back; and when the tumor occupies the descending aorta, it is often louder behind than in front. If, on the back, it has more of the abrupt rasping sound than the ventricular systole in the præcordial region, the evidence of aneurism is almost positive.

3. A purring tremor above the clavicles. I have never found it below, unless the tumor had penetrated through the ribs or sternum. It is weaker than in dilatation, and in old and large aneurisms often becomes extinct.

General signs of sacculated aneurism.—Any or all of the following signs may be present. A pulsating tumor, presenting externally, and sooner or later causing livid redness of the integuments—deficient resonance on percussion; a sense of retraction of the trachea with a wheezing respiration and croaking or whispering voice; dysphagia; an intense gnawing or terebrating pain in the spine; aching of the left shoulder, scapula, neck, axilla, and arm, with numbness, formication, and impaired motive power of the limb; a sense of weight and infarction in the chest; difference of the two pulses; purring tremor of the radials; some of the ordinary symptoms of organic disease of the heart.

Fallacies and the methods of detecting them.—Pulsation beneath the sternum and ribs, occasioned by amplified glands or other tumors in the anterior mediastinum, by hydropericardium, by enlarged heart, or, finally, by adhesion of the pericardium, may, according to my experience, be easily discriminated from aneurismal pulsation by the following criteria.

a. *Pulsating glands* or other tumors in the anterior mediastinum, are not attended with the aneurismal sound; and symptoms of a disturbed circulation either do not exist at all, or do not correspond in severity with the magnitude of the apparent disease.

b. *Hydro-pericardium,* instead of producing the gradual, steady, and powerful heaving of an aneurism, occasions an undulating motion, of which some of the shocks are stronger than others, and none are exactly synchronous with the sound of the ventricular systole. The motion varies its situation, and is occasionally felt over every part of the space occupied by the fluid; whereas in aneurism, the impulse is notably stronger on the tumor and on the heart, than in the intermediate space. Hydropericardium is not productive of the aneurismal sound. Its history is different from that of aneurism, the latter being very often referred to some injury, or excessive exertion, suddenly followed by pain, and dyspnœa.

c. *An enlarged heart* causes a pulsation over a preternatural extent in every direction; an aneurism occasions it in its own direction alone. The beating of an enlarged heart is strongest at the point nearest to the centre of motion, and it decreases progressively on receding from that centre: the beating of an aneurism is stronger on the tumor than at some point intermediate between it and the heart; and in most instances it is stronger even than the beating of the heart itself. Hence, an aneurism distinctly conveys the impression of there being two centres of motion—the tumor and the heart; while the pulsation of an enlarged heart is felt to be referable to one alone. Finally, the ventricular contraction of a dilated heart produces a loud flapping sound, and is not attended with aneurismal murmur or pulsation above the clavicles. I have never known adhesion of the pericardium to occasion a pulsation which could be mistaken for an aneurism, until it had occasioned enlargement of the heart, its ordinary consequence. In this case the diagnostic symptoms are the same as those of enlargement of the heart, with one difference, that the motion is of a more unsteady, jogging, and struggling character.

d. *Varix of the jugular vein*, occasioning pulsation above the clavicle, is distinguished by the absence of sound, the compressibility of the tumor, and the languor of the impulse.

e. *Enlarged glands*, or other tumors, above the clavicles, receiving pulsation from a subjacent artery, rarely occasion sound; and if any exist, it is a feeble whizzing. Both it and the pulsation are confined to the side affected.

If the tumor can be grasped, it will be felt not to dilate laterally during the ventricular contraction, and if it can be raised from the subjacent artery, its beating will cease entirely.

f. *Subclavian and carotid aneurisms* occasion pulsation, sound, and purring tremor on the affected side only, and these signs are more superficial and distinct than in aneurism of the aorta. The sound resembles that of a small hand bellows, instead of having the hoarseness of a forge bellows.

g. *Purring tremor* of the chest, proceeding from mucous rattle, may be recognized by its ceasing when respiration is suspended.

Sacculated aneurism of the abdominal aorta.

This is comparatively so easy of detection, that I have not thought it necessary to enter into much detail respecting its signs.

Physical signs.—1. A constant pulsation of extraordinary power. It appears much stronger to the ear resting on the stethoscope than to the hand. The instrument may be forced down in various directions into close proximity with the tumor, and an idea of its position and dimensions may be thus obtained.

2. A loud, brief, and abrupt bellows sound, not so hoarse as that of aneurisms in the chest. It is sometimes audible on the back. The diastolic sound of the heart is inaudible, and, consequently, the pulsation is simple.

General signs.—They are those of impeded respiration dependent on an imperfect descent of the diaphragm; of lumbar abscess, with or without caries of the vertebræ; of renal disease; and of pressure on the nerves or viscera of the abdomen and pelvis; but none are pathognomonic of aneurism, except a pulsating and usually compressible tumor, felt through the abdominal parietes.

Fallacies and methods of detecting them.

a. *A scirrhous tumor* of the stomach.

b. *Enlargement of the pancreas* by hydatids, or scirrhous—an extremely rare affection.

c. *Fungoid or other tumors* of the mesentery, omentum, transverse arch of the colon, or diaphragm.

d. *Indurated fæces*, air, or masses of tape-worm, impacted in the transverse colon.

When any of these tumors rest upon the aorta, they receive its pulsation, and frequently occasion a bellows sound by compressing the vessel. They may be discriminated from aneurism by their impulse being comparatively feeble, particularly when the stethoscope is applied laterally; by the sound being only a slight whizzing; by the tumor feeling incompressible; by its being superficial when connected with the stomach, colon, or omentum, and by its moving with the movements of these viscera. Finally, the general symptoms are those of dyspepsia, or of malignant disease with slow and progressive marcor, without derangement of the circulation.

e. *An accumulation of serum* in the peritoneal cavity is another source of fallacy, as the liquid transmits both the impulse and the sound of the aorta more distinctly than natural. Dr. Young made this discovery in 1815.* As it is easy to detect the fluid, I have never found this source of fallacy to occasion the least embarrassment.

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* Med. Trans. of Coll. of Phys. of London, vol. v. 1815, No. 15.

Nervous pulsation of the abdominal aorta.—This is a very frequent and deceptive affection in irritable and hysterical constitutions. When it exists in conjunction with air pent up in the colon or duodenum and presenting the feel of a compressible tumor, the resemblance to aneurism is still more complete. After an examination of many cases I am satisfied that attention to the following circumstances will render the diagnosis easy.

Physical signs.—The cylinder may be pressed down on the aorta, so as to yield a distinct feel of the vessel of its natural caliber. The sphere of its pulsation is limited transversely, but extensive longitudinally, being usually more or less perceptible from the epigastrium to the bifurcation. The impulse, instead of being the gradual, steady, and irresistible heaving of an aneurism, is a smart, though vigorous jerk; and the sound, when any exists, is merely a whizzing, almost devoid of hoarseness.

The *general symptoms* are nervous or hysterical, and the pulsation is of an inconstant character, increasing and diminishing with the exacerbations and remissions of the constitutional excitement.

SECTION V.

SPONTANEOUS CURE AND MEDICAL TREATMENT OF ANEURISM OF THE AORTA, AND TREATMENT OF NERVOUS PULSATION.

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Previous to entering upon the treatment of aneurism of the aorta, we shall advert to the mechanism by which its spontaneous cure is effected; as the reader will thus be better enabled to understand the principles on which the treatment is founded.

The movement of the blood within the sac being retarded, partly by the roughness of its internal surface, and partly by the fluid being withdrawn from the direct channel of the circulation, coagulation takes place, and lymph is deposited and organized in successive strata, until the cavity is at length completely filled. The sac being then no longer exposed to the distensive pressure of the circulation, tends to contract by its own resilience and the compression of the incumbent parts, absorption of its contents takes place, and the aneurism is finally reduced to a small, dense, flesh-like tumor. In arteries of the second and inferior orders, the coagulum generally extends to, and obliterates the caliber of the vessel itself;* but this is rarely the case in the aorta, as the force of the circulation in so great a vessel prevents the lodgment of coagula. Instances, however, of obliteration of the aorta by lymph are not without example: an important case has been published by Professor Alexander Monro,† and Dr. Goodison describes another.

It is principally in false aneurism that the cure by deposition of coagula takes place. In true aneurism, and in dilatation, such a cure is very rare; for the walls being unbroken and smooth, and the aperture of communication with the sac being in general large, the blood is seldom arrested to such a degree as to deposit lamellated coagula. When, however, the whole circumference of an artery is converted into a bony cylinder, there is a great tendency to its obliteration by a plug of lymph. Dr. Goodison's case was of this description, and I have more than once seen the same in arteries of the second order.

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* Vid. Hodgson, Jones, Farre, Baillie, Petit, Desault, Scarpa.

† Observations on Aneurism of the Abdom. Aorta by Professor Monro, Ed. p. 5 and 8, 1827.

Hence, as the formation of coagula within the sac is the principal means employed by nature in effecting the cure of aneurisms, the primary object of medical treatment is, to promote the deposition of coagula, and this is best accomplished by such means as have the greatest effect in enfeebling and retarding the circulation. Accordingly, the antiphlogistic treatment, rigorously pursued, is the most efficient remedy for aneurism of the aorta.

This has acquired great celebrity under the designation of the treatment of Albertini and Valsalva. By detraction of blood and spare diet they reduced their patients to so extreme a state of debility that they were scarcely able to raise their arms from the bed. Morgagni reports* that when Valsalva had taken away as much blood as was requisite, he made it a custom to diminish the quantity of meat and drink more and more every day, till he proceeded so far as to allow only half a pound of pudding in the morning, and in the evening half that quantity, and nothing else except water, and this also within a certain weight. After he had sufficiently reduced the patient by this method, so that from weakness he could scarcely raise his hand from the bed, in which he lay by Valsalva's order from the very beginning of the disease, he increased the quantity of aliment by degrees every day until the necessary strength returned.

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* Epist. xvii. art. 30.

To render this treatment safe and efficient, several circumstances must be taken into consideration. In persons of very feeble constitutions, its employment is inadmissible; as it might be fatal by inducing other diseases, or irremediable debility. In individuals sufficiently strong to undergo the treatment, but in whom there is reason to apprehend that extreme debility, if long continued, might occasion pernicious effects, this state, when once induced, should be speedily removed. The depleting system should be actively pursued in the first instance, so as to make a decidedly enfeebling impression on the circulation and thus allow of the contraction of the sac and the deposition of a coagulum; but when the pulse and general feelings of exhaustion indicate that the impression has been made, its protracted continuance should be prevented by a spare but nutritious diet, consisting, for instance, of a little strong beef tea or mutton broth.

The quantity of blood to be drawn must depend upon the constitution of the patient and the effect produced. In one case under my care ℥x had been abstracted daily for sixteen days consecutively with an excellent result. Others I have seen bled to ℥x or xii twice a day for six or seven days and this practice was pursued by Pelletin* and others; but I have found the best effect to be produced with the least expenditure of blood, by drawing a considerable quantity as from ℥xv to xxv in the first instance, and repeating the bleeding to ℥x or xv within twelve hours, and then taking ℥vi or viii every six or eight hours, or at such intervals as to prevent the re-establishment of reaction—a phenomenon which by producing an inordinate energy of the circulation, counteracts the effect of the depletion. Of this I can entertain no doubt, both from extensive observation on the human subject, and from the experiments on dogs described p. 72. In these, the bleedings repeated daily or every second day, occasioned, after three or four abstractions, the most violent arterial throbbing. In individuals who have not sufficient constitutional vigour to give rise to much reaction—less frequent and more sparing detractions of blood will suffice, as, for instance, from ℥vi to xii two, three or four times a week.

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* Clinique Chirurg. tom. i. Prem. Mém. sur les Anevrismes, p. 54.

The blood after repeated abstractions becomes very serous, of a pale crimson instead of the natural dark venous colour, and has sometimes a whitish cream on its surface after standing twelve hours. As such blood contains a very small proportion of fibrine, it is ill adapted for the formation of fibrinous coagula. Hence, this mode of reducing the force of the circulation ought not to be resorted to, if there were any other capable of producing the effect as expeditiously and decidedly. Unfortunately there is none, the operation of hydrogogues, diuretics, &c. being too slow to make an adequate impression in the first instance: but when it has been made, they may be substituted with advantage for blood-letting, as they keep down the quantity of the blood, without depriving it to the same degree of its fibrine. It would not, however, answer to give the patient rich animal food with the view of augmenting the fibrine; for the blood would thus become too stimulant, and, by increasing the energy of the circulation, would defeat the object of the practitioner.

In aneurism of the aorta, especially when conjoined with organic disease of the heart, the bleeding should never, if possible, be carried to syncope; as, under such circumstances, this phenomenon is apt to be alarmingly protracted, and sometimes to terminate fatally. The blood, therefore, should be drawn slowly, and in the recumbent posture. Nor should the venesection be performed during a paroxysm of palpitation, as the exhaustion consequent on it superadded to that occasioned by the loss of blood, not unfrequently sinks the patient beyond the possibility of restoration.

When there is much pain in the tumor leeches afford great relief to it, while they, at the same time, conspire to reduce the circulation; but they should not be applied when the integuments are very thin and discoloured, as they are apt to induce sloughing and rupture of the sac.

Ice, as an application to the tumor, has been strongly recommended, but the pain which it produces is in general intolerable beyond a short time. Its occasional use, however, and, in the intervals, a cold cataplasm of linseed meal and vinegar, are very serviceable by contracting all the tissues and promoting the coagulation of the blood within the sac, when its current has been rendered languid by depletory measures. When cold applications are not employed, and the tumor is painful and requires support, I have found the emplastrum Belladonnæ afford the greatest relief.

The diet should consist principally of fluids, and it should be gradually reduced. Pelletan, sometimes allowed only two basins of broth in twenty-four hours, and lemonade as a common drink. Valsalva, as before stated, gradually reduced the food to half a pound of pudding in the morning and a quarter of a pound in the evening with a limited quantity of water. By thus gradually reducing it both in quantity and quality, the solids may easily he brought as low as four ounces and the fluids as eight, daily. In some even less is sufficient, and this for weeks together. Both the body and the mind should be kept in a state of the most perfect quietude. The recumbent position should be constantly maintained. Purgatives and diuretics, which, as above stated, drain off the serous part of the blood, and have a great effect in weakening the action of the heart, should be frequently administered. Digitalis is useful by producing the same effect, but it should not be pushed so far as to bring the patient strongly under its influence, as the syncope induced by this drug, is liable, in affections of the heart and aorta, to be fatal:—I have several times seen it extremely alarming.

The well known effect of the superacetate of lead in controlling active hæmorrhages, has introduced this as a remedy for aneurism. In Germany it has been extensively used for many years, and Dupnytren, Laennec and Bertin have employed it with advantage in France. My own experience is in its favor. Its tendency to produce colic and inflammation of the mucous membrane of the stomach and intestines may be counteracted by conjoining it with opium, and commencing with a small dose. Half a grain of each, gradually increased to a grain of the acetate in a pill, thrice or four times a day, is the form in which I employ it. Any gastric irritation from it I have always found to be removed by a dose or two of castor oil promptly administered and mucilaginous diluents.

When the patient has been reduced as low as the constitution will bear, the state of the aneurism should be examined with the utmost attention; if it has undergone no amelioration, the treatment should be abandoned rather than the risk incurred of reducing the patient beyond the power of the constitution to sustain. But if it is clear that the pulsation and sound of the aneurism are greatly diminished or entirely suppressed and the volume of the tumor reduced, the practitioner is justified in persevering—so far as he can do it without bringing the life of the patient into palpable danger. When the amelioration has become confirmed, the treatment should be gradually relaxed, but abstinence and the recumbent position should be enforced even for a considerable period after all the symptoms have disappeared.

The treatment of Albertini and Valsalva should not be adopted in a rigorous manner, and with a curative view, unless the practitioner has reason to believe that the aneurism is of the false species, viz. by rupture of the arterial tunics; or, if of the true species, that the sac is not a mere pouch, but so deep, and with so narrow a neck as to be considerably removed from the direct current of the circulation. The latter state may be presumed with some confidence when the tumor, supposing it to spring from the ascending aorta, extends far to either side; or, whatever be its situation, when its base is remote from the caliber of the artery. Aneurisms of the descending aorta may be treated as false, post mortem inspection having proved that they are almost always of that description. In cases of dilatation, and of shallow true aneurism, the antiphlogistic plan should be pursued to a moderate extent only, and merely with a palliative object: for as, under such circumstances, coagula scarcely ever form, a radical cure is not to be expected.

It may be said, finally, that the efficacy of the treatment of Albertini and Valsalva has, in all probability, been somewhat overrated. For, as the diagnosis of aneurisms of the aorta was involved in much obscurity until the last few years, it is certain that many cases reported as cured, were not aneurisms, but tumors or nervous pulsation simulating that disease. Another reason has prevented the treatment from maintaining its ground; namely its severity. Though patients will submit to rest and extreme abstinence, they have rarely fortitude to see blood-letting superadded. The practitioner, on the other hand, has seldom the courage to insist upon it, knowing that it is not wholly exempt from danger and that it will not necessarily be productive of a cure, especially if not pursued with uncompromising rigour.

Treatment of nervous pulsation of the aorta.—Though the treatment of nervous pulsation does not strictly come under the head of aneurism, yet, as the pulsation is a frequent concomitant of aortic aneurisms and organic diseases of the heart, it may not be foreign to our present purpose to advert briefly to its treatment.

The indication is, to allay the nervous irritability and excitement on which the pulsation depends. This may be effected by causing the patient to maintain, as far as possible, a tranquil state of body and mind; by a mild, cooling diet and regular state of the bowels; by sedative remedies, as conium, hyoscyamus, camphor mixture, and, if there be hysterical symptoms, by these conjoined with antispasmodics, as assafœtida, valerian, sulphuric æther. It is scarcely necessary to add that attention to the catamenial secretion is of primary importance. When the patient is sufficiently tranquillized to bear tonics and stimulants, the best remedies are, the various preparations of steel, with infusions and decoctions of cinchona, cascarilla, columba, orange-peel, &c. the shower-bath, much out-door exercise, alternated with rest so as not to fatigue, and a nutritious, but not heating diet.

CHAPTER XI.

MALFORMATIONS OF THE HEART.

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MALFORMATIONS of the heart are imperfections, generally congenital, in the structure of the organ, and they consist in a deficiency, a superabundance, or an anomalous configuration of parts. The number of varieties of malformation is considerable, and they are so irregular in their combinations as scarcely to admit of being classified on general principles. All worthy of notice that have hitherto been met with, are comprised in the following catalogue.

1. The heart is single, like that of a fish, consisting of one auricle, and one ventricle from which springs a trunk that presently divides into the aorta and pulmonary artery. The patients have generally died within ten days.*
2. There are two auricles, and one ventricle. In one case the patient attained the age of twenty-two.†
3. The foramen ovale remains open. This is the most common malformation, and is found at all ages, sometimes even at the extreme period of senility.‡

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* Vid. a Case in the Philos. Trans. v. 88. p. 346—another, ibid. v. 95. p. 228—another in Dr. Farre's Path. Research. Essay 1. p. 2.—and two in the Ephem. nat. cur. Dec. 1. ann. 4 and 5, Obs. 40—and Dec. 2. ann. 10, Obs. 44.

† Case by Wolf, mentioned by Kreysig, die Krankeitendes Hertzens. Berlin, de 1814 à 1817. v. iii. p. 200—and one was seen by Breschet.

‡ Passim. It has been found in the aged by Albinus. Academ. Annot. Lib. i. Cap. ix. and Burns, on Diseases of the Heart, p. 8.

4. The foramen ovale and ductus arteriosus both remain open.*
5. The foramen ovale and ductus arteriosus are open, and the pulmonary artery obliterated at its origin. In one case, the cavity of the right ventricle was nearly obliterated, and, in two others the septum of the ventricles was perforated.†
6. The septum of the ventricles is totally deficient, and that of the auricles very imperfect.‡
7. The aorta arises from both ventricles, i. e. The septum of the ventricles being deficient at the mouth of the aorta, forms a common opening between that vessel and the two ventricles. It is generally accompanied with contraction of the pulmonary artery, frequently with an open state of the foramen ovale, and occasionally with obliteration of the pulmonary artery and patescence of the ductus arteriosus.§

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* Deschamps, Fouquier, Thibert, Monro, Burns, &c.

† W. Hunter, Med. Obs. and Inq. v. vi. p. 291.—Farre, two Cases, Path. Research. p. 19. Two died within thirteen days: one lived six months.

‡ Farre, Path. Research. p. 30. Senac, Traité sur la Structure du Cœur. v. ii. p. 404.

§ Corvisart. p. 293-8, three Cases.—Sandifort, Obs. Anat. Path. cap. 1. p. 35.—Bartholinum, Acta Hofniensia, tom. i.

8. The septum of the ventricles is perforated. The aperture is small, and, though near, it is not immediately in, the mouth of the aorta. With this state, the pulmonary artery is sometimes contracted and the foramen ovale open.* A similar perforation appears to be formed by ulceration, and this, in one case, took place at the point of junction of the septum of the auricles and ventricles, so that the four cavities of the heart communicated.†
9. The pulmonary artery arises from both ventricles and the foramen ovale is open. This vessel sends off the *descending* aorta, while the *ascending* arises in the natural way.‡
10. The aorta springs from the right ventricle, and the pulmonary artery from the left, the foramen ovale, and sometimes also the ductus arteriosus, remaining open.§
11. The right auricle opens into the left ventricle instead of into the right, and the ventricles

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p. 200.—Abernethy, Surg. and Phys. Essays.—Farre, Path. Res. p. 26.—Ed. Med. and Surg. Jour. v. ix. p. 399. Tredeman, Stander. The Writer, p. 468.

* Dr. Hunter, Med. Obs. and Inq. v. vi. p. 299, two Cases. —Corvisart. p. 276.

† Laennec, t. ii. p. 547.—Thibert.

‡ Two Cases by Sir A. Cooper.

§ Farre, Path. Research. p. 29.—Langstaff, Lond. Med. Rev. p. 88.—Baillie, Morbid Anat.

communicate by an aperture immediately below the aortic valves. The foramen ovale is open.*

12. The arch of the aorta was double in a child of twelve or thirteen years old seen by Bertin the father.
13. The foramen ovale is closed in the fœtus.†
14. The valves sometimes exhibit defects which appear to be congenital. The mitral, the tricuspid, and the pulmonic valves have been found stretched flat across their orifices, with a perforation in the centre;‡ and the membranous part of the several valves has been found perforated: in one instance it resembled a net-work.§

Of all the causes of communication between the two sides of the heart, patescence of the foramen ovale is the most frequent. This either results from the two layers of which the valve consists in the fœtus, not becoming adherent,—a common form of patescence and one which does not appear to occasion any material inconvenience: or the foramen is dilated and permanently open, being sometimes large enough to admit the thumb. This dilated state is generally supposed to be congenital; but, as many patients have dated their symptoms of disease of the heart from a fall, blow, or violent effort, it is probable that these accidents had caused either the rupture of the membrane closing the foramen, or the separation of its imperfectly agglutinated layers; whence ensued the progressive enlargement of the aperture.

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* A case by Holmes, Ed. Med. Chirurg. Trans. p. 252. The right auricle equalled a pint in capacity. The patient attained the age of twenty-one.

† Vieussens, sur la Structure du Cœur, c. viii. p. 35.

‡ Burns, Morgagni, Laennec, Louis, Bertin.

§ Laennec, ii. p. 550.

Whatever be the mode of communication between the two sides of the heart, its effect is, with few exceptions, to cause an intermixture of the arterial and venous blood. One exception, and the most common, is, when the two layers of the foramen ovale are simply non-adherent; in which case the pressure of the blood on each side closes them like a valve—a pressure which exists as well during the diastole, as the systole of the auricles; for, according to the experiments of the writer, the auricles are constantly *full*, though sometimes more distended than at others. A second exception may possibly exist when the pressure of blood on each side of the gaping aperture, is equal. Even under these circumstances, however, it is probable that there would be a slight intermixture. But such cases are, I believe, more imaginary than real; for it scarcely ever happens that there is not, on one side or the other, some valvular or analogous obstruction, which, by impeding the current of the blood along its natural channel, renders its pressure through the morbid aperture stronger than that in the opposite direction. Thus, in more than half the cases of communication between the right and left cavities of the heart, there is a contraction of the pulmonary orifice, or of the pulmonary artery itself.

This brings us to the fact, that the communication of the two sides of the heart is almost constantly accompanied with hypertrophy or dilatation of the right cavities, whereas the left are very rarely affected. M. Bertin attributes this to the introduction of a certain quantity of red, *arterialized* blood into the right cavities, which he thinks calculated to occasion their hypertrophy in consequence of its being more irritating, more nutritive, possessed of more vitality, than the venous blood.

I doubt whether this ingenious hypothesis is tenable, as the most remarkable cases of hypertrophy of the right ventricle have been those in which there was extreme contraction of the pulmonary orifice, when, consequently, the current through the foramen ovale must have been so decidedly from the right to the left side, that no arterial blood could possibly have entered the right ventricle.

What, then, was the cause of the hypertrophy of that ventricle? The contraction, I should imagine, of its pulmonary orifice; in the same way that contraction of the aortic orifice occasions hypertrophy of the left ventricle. M. Bertin supports his opinion by the circumstance that the hypertrophy is often accompanied with contraction of the cavity, which, he thinks, would not be the case if the hypertrophy resulted merely from too great a quantity or two great a distending pressure of the blood. To this it may be replied that, in the left ventricle, hypertrophy with contraction arises more frequently from a similar cause, that is to say, obstruction of the aortic orifice, than from any other; the reason of which I have attempted to explain in the article on hypertrophy, p. 199. It might be objected that, in many cases, though the pulmonary artery was obstructed, the ventricle discharged itself by an opening into the left ventricle or into the aorta. True; but this discharge was not made with the same facility as in the natural way through the pulmonary artery, inasmuch as the weight of the aortic circulation exceeds that of the pulmonary.

Having said so much to account for the hypertrophy, we have next to consider the cause of the dilatation which is occasionally found in the right cavities. This is manifestly an effect of over-distention; for, as far as I can discover, it is always accompanied with an excess of blood on the right side, determined there in consequence of some obstruction to its course on the left. Thus, in a case by Corvisart,[*] the excessive smallness of the aorta caused the blood to flow through the foramen ovale, which was more than an inch in diameter, and thus to produce dilatation with hypertrophy of the right cavities.

While I thus contend that there are sufficient *mechanical* causes to account for hypertrophy and dilatation of the right cavities in cases of communication between the two sides, I do not wish to assert that the introduction of arterial blood may not contribute to the production of hypertrophy. On the contrary, I think it probable that it does, since the arterial is a morbid stimulus of the right cavities; but it is repugnant to the principles of inductive science to assign this, which is at best problematical, as the sole cause, overlooking others, the effect of which is unquestionable.

General signs of communication between the two sides of the heart.—The signs given by authors, are, a violet or blue colour of the skin, in general much more intense and extensive than in any other malady, and sometimes even universal; a reduction of temperature with great sensibility to cold; unusually frequent attacks of syncope; occasionally convulsions; and a greater difficulty of respiration than in most other diseases of the heart.

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[*] Sur les Maladies du Cœur, p. 279.

Of the blue discoloration of the skin, which has been designated by the names *blue disease*, *blue jaundice*, *cyanosis*, it may be said, that, when the intermixture of the arterial and venous blood is not very considerable, and especially when the admission of venous blood into the lungs is free, the discoloration is sometimes not deeper than is to be found in cases of ordinary obstruction to the return of the venous blood, and occasionally it scarcely exists at all. On the contrary, when the ingress of venous blood into the lungs is very limited and the intermixture with the arterial considerable, the colour is of the deepest dye, and pervades not only the lips, nose, ears, and face, but the hands, the feet, and, in greater or less intensity, the skin universally. Such, at least, is the generalization to which I have been brought by an examination of nearly all the cases that have been published on this subject.

According to M. Laennec, the blue colour of the skin is equally marked and extensive in some diseases of the lungs, particularly emphysema, as in cases of communication between the two sides of the heart. This is not consistent with my own observation, and it is, I presume, very rare; for of many thousand cases of pulmonary disease which I have seen, in not one, nor in any ordinary organic disease of the heart, has the colour admitted of comparison with that which I have witnessed, and of which I shall presently offer an instance, in cases of the communication in question.

For this reason I must dissent from the opinion of M. Bertin, who maintains that the blue or violet colour depends, not on intermixture of the black with the florid blood, but, simply, on the same cause that occasions it in cases of ordinary obstruction to the circulation: namely, "the stagnation of the blood in the right cavities of the heart, and in the venous system, which is as it were gorged with it." Were this true, cases of intense discoloration would be of ordinary occurrence, instead of being extremely rare and presenting themselves in those almost exclusively who are affected with a communication between the two sides of the heart.

It is scarcely necessary to remark, that when hypertrophy or dilatation co-exists with malformation, the effects resulting from them are added to those of the congenital disease.

Physical signs.—Laennec had not an opportunity of studying cases of malformation with the stethoscope. In one case, of which I made a post-mortem examination, the signs were conformable to the general principles developed in the several articles on organic diseases of the heart: namely, enlargement of the organ by one half was indicated by dulness of the præcordial region on percussion and slight prominence; hypertrophy of the right ventricle was denoted by increased impulse at the inferior part of the sternum; contraction of the pulmonary orifice and a common opening of the right ventricle with the left into the aorta, occasioned a loud, *superficial* hissing murmur with the first sound, loudest about the middle of the sternum, over the orifices affected.

In another patient, still living, the symptoms are almost identical. Whether patescence of the foramen ovale alone would occasion a bellows-murmur, I cannot say from experience; but from analogy I am inclined to think that it would, provided one side of the heart were obstructed. For during the ventricular diastole, when a greater quantity of blood passes from the auricles into the ventricles than at any other time, there would be a current through the foramen ovale towards that side of the heart which received and transmitted the blood most freely; and such a current, if sufficiently strong, would excite a murmur *synchronous with the second sound.* But, supposing no obstruction to exist, and that both ventricles received and transmitted the blood with equal freedom, there would be no appreciable current through the foramen, and consequently no murmur.

Cases.

The two following cases present excellent exemplifications of cyanosis. The history of the latter was drawn up, at my request, by the father, a gentleman of great intelligence and observation. It is valuable as displaying the habitudes of a patient affected with this disease in a much more graphic manner than can be done by a mere enumeration of symptoms.

Mary Collins, æt. 8, applied to me October 22th, 1830. Lips, nose, cheeks, palpebræ, hands and feet of a violet colour: tongue and mouth still darker. On a frosty day, after walking or ascending stairs, the hue of the parts enumerated, as witnessed by myself and several medical friends, is equal to the deep stain communicated to the skin by black currants or the small black cherry, and the face and hands universally are as dark as those of a mulatto. Children in the streets often enquire in winter, "where she got blackberries at that season." Dyspnœa on the slightest exertion, particularly ascending; cough when hurried, not otherwise; sternum very prominent; great sensibility to cold—constantly steals to the fire, even in summer; headache, vertigo, drowsiness, and sluggishness. Pulse, very small and weak, and when hurried, it is irregular, intermittent and unequal.

Auscultation.—Resonance of the præcordial region dull. *Impulse*, considerably stronger and more extensive than natural—strongest over the right ventricle. *Sounds.* The first, a very loud, hissing, superficial bellows-murmur: the second, natural or nearly so. In six months she died of a chronic abscess in the brain.

Diagnosis. Patescence of the foramen ovale: hypertrophy of the right ventricle; obstruction of the orifice of the pulmonary artery. (Stated before the dissection, at which Dr. Marshal Hall, Mr. Else, surgeon, and others were present.)

Sectio.—The heart was one half larger than natural: the walls of the right ventricle were thickened to half an inch: the cavity was slightly dilated and its pulmonary orifice contracted to the size of a goose-quill, while a common opening from the right ventricle admitting the index finger, existed into the aorta and the left ventricle. The left ventricle was one third of an inch thick, and its cavity about natural. The two layers of the foramen ovale were disunited and the handle of a large scalpel easily passed obliquely through them. The lungs were rather flaccid, imperfectly crepitant, and universally gorged with black blood.

In this case, it was the *superficial* nature of the murmur which led me to conceive that it was occasioned by an obstacle to the passage of blood from the right, rather than from the left ventricle.

Master R. æt. 11. While he is tranquil and warm, the complexion is about two shades darker than natural and its tint is a purplish crimson: on the lips the colour is deeper, and within the mouth it is a blackish violet. On ascending a flight of stairs the colours become intense. The hands and feet are of the same hue, and the last phalanges of the fingers and toes are bulbous, being one third larger in circumference than the phalanges above. Pulsation of the carotids: engorgement, without pulsation, of all the veins of the neck. Left margin of the sternum prominent opposite to the fifth, sixth, and seventh ribs. *Resonance* deficient over the whole præcordial region. *Impulse* much increased, strongest where the sternum is prominent. *Sounds.*—The first is a loud, prolonged, bellows-murmur, loudest opposite to the arterial orifices of the heart: the second is short and loud, with a very slight filing murmur.

The following history is given by the father. "He was always as blue as at present, and I think more so when very young. Until the age of nine months, he was very subject to spasms of the bowels, and also, as I used to think, of the chest.

They were in general relieved by immersion of the lower extremities in warm water, (which was always kept so as to be ready within two minutes,) and by a dose of castor oil in anise water. His respiration was always quick, and he was always subject to cough, particularly on taking cold, to which he is very liable. He perspires very freely, and, about the hands and feet, to a great degree. When the weather is sharp and nipping, he becomes exceedingly blue. He is very chilly and sensible to cold. When exposed to a cold, or damp and cold atmosphere, he becomes as it were *asthmatic;* his corporeal powers are overcome with numbness, he loses, in a great measure, the faculty of motion, and I am sure that, if placed, on a cold day particularly with an east wind, in an exposed situation two miles from home, and left to return that distance by his own exertions, his powers would become so deadened that he would perish in the attempt. I have sometimes, under such circumstances, been obliged to bring him home in a coach or even in my arms. He suffers less in severe *frosty* weather, than when the wind blows cold and harsh though the temperature be six or eight degrees above the freezing point. However much inconvenienced by exposure to cold, he regains all his powers by the time that he has remained half an hour in a warm room. In mild weather he is less blue, and his respiration less oppressed. If he cuts or scratches himself, he bleeds more than others. The finger and toe-nails are scarcely thicker than paper, and they grow very fast, requiring to be cut every four or five days. He is very subject to cynanche tonsillaris with great swelling of the parts. He suffers more than other children from illness produced by slight or common causes, and his health consequently suffers frequent interruptions. I must remark, in particular, that when he becomes ill, it is not gradually, by the progressive developement of the symptoms, but suddenly, scarcely ever giving the slightest premonitory signs. Not half an hour before a severe attack, he has often appeared in good health. He grows remarkably fast. His temper is very quick and irritable, but his disposition is candid, frank and generous: his mind is active and ardent. Tongue never quite clean and the papillæ are very large: appetite and digestion generally good: bowels free: urine almost always turbid.

PART IV.

NERVOUS AFFECTIONS OF THE HEART.

THE nerves of the heart, as of every other organ, may be affected in two ways. They may labour under over-excitement, dependent either on increased irritability or on excessive stimulation; and they may be in a state of deficient excitement, dependent either on diminished irritability or on inadequate stimulation. These states, when existing in a moderate degree, cannot strictly be considered morbid. Thus, palpitation from exercise or from an exhilarating mental emotion, and languor of the heart's action from a depressing passion, do not rank as diseases. But when the states in question exist in excess, and when they result, less from remote sympathies, than from a primitive affection of the nerves of the heart itself, they constitute diseases. Of these, the state of over-excitement comprises *neuralgia of the heart or angina pectoris*, and *palpitation, spasm, and convulsion of the organ:* while the state of deficient excitement presents *syncope*. These diseases will be considered in successive chapters.

CHAPTER I.

NEURALGIA OF THE HEART OR ANGINA PECTORIS.

Symptoms of Angina Pectoris, 474. *Causes of the Attack*, 476. *Its Duration if the Disease is Acute*, 476. *If Chronic*, 476. *Chronic generally connected with organic Disease of the Heart*, 476. *Discordant Opinions of Authors respecting the predisposing Cause of Angina*, 476. *Irritation or morbid Irritability its real Cause*, 477. *Organic Disease of the Heart the most frequent Cause of the Irritation*, 477. *Exemplified and explained*, 478. *Pulse sometimes feeble and irregular, why*, 478. *Aggravated Angina perhaps never independent of Disease of the Heart*, 478. *Slight Angina may be*, 479. *A common Affection*, 479. *Under what Circumstances*, 479. *How occasioned by abdominal Tumors*, 479. *Nerves in which the Pain originates and those by which it is propagated*, 480. *Evidence that Angina is a neuralgic Affection*, 480. *Prognosis*, 481. *Treatment*, 481. *Carminatives, Antacids, Emetics, Blood-letting, Antispasmodics and Sedatives*, 481. *Treatment during the Intervals*, 482. *For Chronic Angina, Counter-irritants, &c.* 482. *Treatment when conjoined with Hysteria, Dyspepsia, &c.* 483. *Magnet as used by Laennec*, 483.

Neuralgia of the heart occurs in paroxysms of greater or less severity and at longer or shorter intervals. When presenting the train of symptoms which have been denominated by Dr. Heberden *angina pectoris*, it commences by a sensation of pain and constriction in the præcordial region, accompanied with a more or less painful numbness in the left arm, more rarely in both arms, still more rarely in the right arm alone, and occasionally in all four extremities,—of which I have, at present, a case under my care.

At first the pain may not reach beyond the insertion of the deltoid muscle, but it soon extends down the inside of the arm to the elbow, and sometimes accompanies the ulnar nerve to the extremities of the fingers. It is not unusual for pain to exist at the same time in the left anterior part of the chest, following the tract of the anterior thoracic nerves, and occasioning in females so great an increase of the sensibility of the mamma, that the slightest pressure becomes painful. When the attack is smart or what is called *acute*, the pain is excruciating, appearing to the patient as if "iron nails or the claw of an animal tore asunder the anterior part of his chest."* With this, there is great pulmonary oppression, amounting, in the worst cases, to suffocative orthopnœa; the heart either palpitates violently, or it falters, flutters and intermits;† congestion of blood in the head, syncope and convulsions sometimes ensue.

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* Laennec de l'Auscult. tom. ii. p. 706.

† I suspect that some authors, who have described the

The attack is commonly induced by some over-excitement of the heart, especially that of walking up hill, to the effect of which a recently loaded stomach and a wind in front, powerfully contribute. The patient is compelled instantly to stop, and, if the complaint be recent, the attack sometimes subsides in the course of a few minutes by mere rest, and seldom continues longer than from half an hour to an hour, even in cases so severe as to prove fatal. Should the patient survive the first attacks, the disease is apt to become chronic;—when it recurs more frequently, is excited by slighter causes, and is sometimes so obstinate as to resist every remedy for several hours and even for as many days. In these cases it is seldom that there is not some irregularity or other peculiarity of the pulse denoting organic disease of the heart, and that the latter is not found on post-mortem examination.

Causes of angina pectoris.—Great diversity of opinion has existed respecting the cause of angina pectoris. Different physicians have found it connected with different organic lesions or states, and each has supposed it to be occasioned by that,

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pulse as calm, have mistaken these characters for calmness. I have never seen the action of the heart undisturbed in a severe case, nor do I think it possible.

with which he has most frequently found it coexist. Dr. Parry, and after him Burns and Kreysig, ascribe it to ossification of the coronary arteries; Dr. Hooper to affections of the pericardium; Dr. Hosack, to plethora; Dr. Darwin, to asthmatic cramp of the diaphragm; Drs. Butler, Macqueen, and many others, have regarded it as a particular species of gout; Dr. Latham has found it connected with enlargements of the abdominal viscera while the thoracic viscera were sound; and Heberden, having found it both connected and unconnected with organic disease, thinks that its cause has not been traced out, but that it does not seem to originate *necessarily* in any structural derangement of the organ affected.

They who have ascribed angina pectoris to any particular cause to the exclusion of others, have unquestionably taken too limited a view of the subject; as experience has fully proved that it may originate in various causes. According to my own observation, it may originate in any cause, whether organic or functional, capable of *irritating* the heart, or of rendering it morbidly susceptible of irritation; and as structural disease of the organ has this effect more than any other cause, it is that on which the malady is most frequently dependent.

The most violent cases of angina that have occurred to myself, and, if I mistake not, that have been recorded in books, have been connected with osseous, cartilaginous, steatomatous or other degeneration of the heart or great vessels, by which some portion of them, especially the coronary arteries, the valves and the commencement of the aorta, was more or less deprived of its elasticity. Hence it may be perhaps reasonably conjectured that, when the action of the organ is excited, as by ascending a hill, a loaded stomach, &c. the over-tension of the rigid portion is the source of the irritation and pain. It is no objection to this view that, at the time when the pain is the most intense, the action of the heart is sometimes diminished—that it feebly flutters and falters, and that the pulse has the same characters; for, so long as these phenomena display themselves, the heart is in a state of engorgement—of even greater distention, perhaps, than when it is acting violently. The proofs that such is the case are manifest in the suffocative orthopnœa, the tumid, livid state of the face, and the diminution of the sounds of the heart.

I have also several times seen angina of considerable, but not equal severity, accompany hypertrophy and dilatation with or without softening; but I have never known the malady to exist *in an aggravated form*—one which truly merited the name of *angina* rather than of mere neuralgia, independent of *some* organic disease of the heart or its immediate appendages. Cases, it is true, are on record which appear to militate against this opinion; but, as it is only of late years that the anatomical characters of hypertrophy, of dilatation, and of softening have been clearly understood, the evidence of such cases must be admitted with reserve. To mention an instance,—a case of sudden death from angina said to be independent of disease of the heart, was recently communicated to me; but, from the statements of the parties present at the dissection, it was clear that there existed a marked dilatation, which they had not recognized.

Angina, however, in a moderate degree may, as Laennec maintains, exist independent of any organic disease of the heart or great vessels, and it is, indeed, a very common affection. I have frequently met with it in hysterical females subject to palpitation and in cases of nervous dyspepsia and hypochondriasis, under the form of spasmodic, aching pains in the anterior part of the chest, extending sometimes to the neck and stomach, and either attended or not with pain and numbness in one or both arms. One of my medical friends always feels the affection of the arm when attacked with dyspeptic palpitation, to which he is subject.

It is very conceivable that, if the irritation of a loaded and dyspeptic stomach can create angina, an enlarged liver or other abdominal tumor, by displacing the diaphragm, or by mere sympathetic irritation, might, as in the cases of Dr. Latham, produce the same effect.

Nerves affected in angina—M. Desportes places the seat of angina in the pneumo-gastric nerve or par vagum, because the lungs, as well as the heart, are affected with pain and have their function disturbed. Laennec thinks, with great reason, that the filaments which the heart derives from the sympathetic, are likewise implicated in the disease; because there is sometimes pain in the organ without any in the lungs or material embarrassment of the respiration. From these nerves the irritation is propagated, either by sympathy or by anastomosis, to others; namely, to the superficial cervical plexus and its anterior thoracic branches, whence proceeds the pain in the neck and in the surface of the chest; to the branches of the brachial plexus, especially the ulnar, whence arises the pain descending to the elbow and sometimes to the fingers; finally, to the branches of the lumbar and sacral plexus, whence the pain and numbness felt in the thighs and legs, and even in the spermatic cord and testicles.

The nature and variability of the symptoms of angina pectoris, confirm the opinion of Laennec that it is a neuralgic affection; for those neuralgic affections whose nature is least equivocal,—sciatic gout or tic douloureux, for instance, produce, in different degrees, effects of the same nature and equally diversified as those of angina pectoris; that is to say, acute pain, painful torpor, simple numbness in the tract of the affected nerve, and sometimes spasm or sub-inflammatory intumescence of the parts to which the nerve is distributed.

Prognosis.—When the malady is dependent on organic disease of the heart or great vessels, the prognosis is decidedly unfavourable; for in addition to the danger which always attends the organic disease in question, there is that of fatal syncope from the angina—a termination to which this affection is prone, and which rarely occurs, even in the worst cases of disease of the heart, independent of angina. When the complaint is symptomatic of dyspepsia, hysteria, &c. the prognosis is favorable.

Treatment.—When angina depends on organic disease of the heart, it must be treated on the general principles which regulate the treatment of the latter. The patient should instantly be placed in a state of repose; flatus of the stomach, if present, should be extricated by a draught of peppermint-water with anise oil, sp. ætheris sulph. and aromatic confection: acidity should be neutralized by a free dose of soda or prepared chalk; and, if the stomach be loaded with an irritating mass of undigested food, it should be evacuated by ipecacuan with sulphate of copper or of zinc, provided the state of the respiration be such as to admit of the effort of vomiting. Should the distress be extreme and the patient plethoric, six, eight, or ten ounces of blood may be drawn, and, for this purpose, cupping or leeches on the præcordial region, if the patient is sufficiently tranquil to admit of them, often affords more relief than venesection.

These preliminary measures having been carried into effect as expeditiously as possible, an antispasmodic and sedative draught should be administered. It may comprise a full dose of tinct. or extr. opii, or, as less exciting, of the liquor opii sedativus, or of acetate of morphia, or two drops of hydrocyanic acid, with sp. ætheris sulph. comp. ʒss and mist. camph. or solut. assafœtid. ʒx. The draught and the other measures must be repeated according to existing circumstances, of which the practitioner is the only judge.

During the intervals of the fits, the general health must be improved and the recurrence of the fit prevented by the same means as in organic disease of the heart. (See Dilatation, p. 282.)

When the complaint is chronic, counter-irritants and derivatives, as blisters on the chest, sinapisms on the legs, and setons or issues in the thighs, have been found useful. Nature seems, indeed, to indicate these remedies; for, where the disease has not been dependent on organic causes, it has often yielded to some natural counter-irritation or vicarious discharge, as a hæmorrhoidal flux of blood or serum, an obstinate gleet, &c.

In cases dependent on hysteria, dyspepsia, &c. the primary malady demands the first attention, while the neuralgic pain may be combated by counter-irritants and occasional sedatives. The metallic tonics, especially the carbonate of iron in doses of from ℈ij to ʒi thrice a day, are the most efficacious internal remedies for it.

The remedy by which Laennec states that he has most frequently succeeded in procuring alleviation in cases of angina pectoris and of neuralgia of the heart of a slighter kind and without radiating pain, is the magnet, which he employs in the following manner: he applies two steel plates strongly magnetized, of a line in thickness, of an oval form, and slightly arched so as to apply closely to the thoracic walls, the one on the left præcordial region, and the other on the opposite part of the back, in such a manner that the poles may be exactly opposite and the magnetic current may traverse the part affected. This remedy, Laennec adds, is fallible no less than all those by which we ordinarily combat nervous affections; but it has succeeded in his hands oftener and to a greater extent than any other. When it procures little alleviation in angina, more may sometimes be obtained by applying a small blister under the anterior plate. It should be remarked that they who witnessed the application of the magnet by Laennec, did not, in general, form so favourable an opinion of its utility as that author himself. It appeared to be more successful when combined with acupunctuation.

CHAPTER II.

PALPITATION, PARTICULARLY NERVOUS.

Varieties of Palpitation depend on Differences in their Causes, 485. *How these Causes operate*, 486. *Palpitation defined and described*, 486. *Palpitation from mere Acceleration of the Circulation*, 487. *From Hypertrophy, from Hypertrophy with Dilatation*, 487. *From Dilatation with Attenuation*, 487.

NERVOUS PALPITATION, *described*, 488. *The Impulse of*, 489. *Diagnosis from Disease of the Heart*, 490. *Signs derived from Dyspepsia not pathognomonic*, 490. Physical Signs. *Impulse*, 491. *Murmurs*, 492. Cases. *First*, 493. *Second*, 495. *Third*, 497.

AFTER presenting a general view of the nature and causes of palpitation and adverting briefly to its varieties, I shall dwell more particularly on that which is purely nervous.

As palpitation is, under all circumstances, dependent on over excitement of the nerves of the heart, the phenomenon, in its essential nature, is always the same. The varieties which it presents arise merely from differences in their causes, and from the different routes which these causes pursue in order to arrive at, and convey their stimulus to the heart. Thus the blood conveys the stimulus, *directly*, and in three ways; first, by arriving in excess, as from violent exercise, plethora, &c.:* secondly, by gorging the heart in consequence of its transmission being impeded by a disease of the organ, or an obstacle in some other part of the circulation; thirdly, by being of too stimulant a nature, in consequence of the diet being exciting. The nerves, on the contrary, convey the stimulus to the cardiac plexus *indirectly*, as is the case in emotions of the mind, in dyspepsia, in hysteria, &c. The nerves and the blood may also convey the stimulus conjointly, as probably happens in fever.

Palpitation in general may be defined to be an increase in either the force or the frequency, or in both the force and frequency, of the heart's contractions, by which they become not only sensible, but sometimes very troublesome to the patient. They may vary in force from a scarcely perceptible degree, to a violence which amounts to convulsion. Not unfrequently the sound of the beats is audible to the patient, especially when lying on his side; and in this position, the second as well as the first sound may occasionally be perceived.

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* I suspect that palpitation from excessive loss of blood will come under this head; for, though the quantity of blood be diminished, its remarkable attenuation enables it to traverse the vessels with greater facility; and it probably, therefore, arrives at the heart either in redundant quantity or with morbid velocity. This view is perhaps more consistent than the anomalous one of palpitation being produced, in cases of loss of blood, by a deficiency of the natural stimulus: an explanation which some have offered.

When the circulation is simply accelerated, as by exercise, &c. in a healthy subject, the palpitation consists in an increase both of the force and the frequency of the heart's action. The same occurs in hypertrophy, and hypertrophy with dilatation, and the physical characters of the impulse and sounds are merely an exaggeration of those which the heart presents during a state of calm. (Vid. hypertrophy and hypertrophy with dilatation). In dilatation with attenuation, palpitation sometimes consists in an increase of the frequency, but not of the strength of the beats, though the patient may experience the sensation of an increased impulse. Palpitation of this kind is more obstinate than any other. Laennec cites an instance in which it lasted eight days, the pulse constantly beating 160 to 180 per minute.

It must be recollected that, in every organic disease of the heart, when palpitation becomes extremely violent and prolonged, both the impulse and the sounds may be diminished—in other words the heart becomes gorged and incapable of adequately contracting on its contents, sometimes yielding a struggling convulsive impulse, with little sound and a feeble pulse, and, in an ulterior degree, especially during dissolution, scarcely producing either impulse, sound, or pulse.

Nervous palpitation imitating disease of the heart.—There are few affections which excite more alarm and anxiety in the mind of the patient than this. He fancies himself doomed to become a martyr to organic disease of the heart, of the horrors of which he has an exaggerated idea; and it is the more difficult to divest him of this impression, because the nervous state which gives rise to his complaint, imparts a fanciful, gloomy and desponding tone to his imagination. Members of the medical profession are more apt than others to give way to these feelings; partly from their apprehensions being more keen, and partly from an impression too widely prevalent, that there is difficulty in distinguishing nervous from organic palpitation, and, consequently, that they must remain in a state of anxious uncertainty. It may be said for the consolation of such, that the diagnosis presents no difficulty to one who, to general signs, adds a knowledge of those afforded by auscultation and percussion.

Nervous palpitation, dependent on dyspepsia, hypochondriasis, hysteria, gout, mental perturbation, &c. presents very diversified characters. The slightest degree of it I should describe, from having occasionally experienced it, to be a tumbling or rolling motion of the heart, with a momentary feeling of tightness and oppression. It is referable to an intermission of the heart's action. In a further degree, there is a series of quick, weak, fluttering irregular beats with slight anxiety, acceleration of the respiration and a quivering sensation in the epigastrium: this may last from a few minutes to half an hour or an hour, and occur only at distant and irregular intervals, or repeatedly during the day, especially when the patient is startled. The next degree amounts to a perfect fit of palpitation consisting in increased impulse, sound and frequency of the beats, sometimes with irregularity, and generally with more or less anxiety, dyspnœa, and even orthopnœa. The attack may be only occasional, or may occur several times a day, or may even last with little intermission for several days together. Sometimes a degree of palpitation subsists for years, especially in young persons of a constitution at once plethoric and nervous.

In nervous palpitation, the impulse is less remarkable for force, than for an abrupt, bounding character, with throbbing of the arteries, often universal, and a jerking pulse. Hence this species of palpitation is more audible to the patient than perhaps any other, the sound appearing to rush through his ears, especially when he lies on his side in bed and each arterial throb causes a movement of his pillow.

Nervous palpitation may be distinguished from that of disease of the heart by the palpitation occurring only occasionally: by its not being excited, but, on the contrary, relieved by corporeal exercise of such a nature as would certainly disturb the action of a diseased heart: by its disposition to supervene while the patient is at rest, especially at the commencement of the night when he lies wakeful in bed; by a fluttering in the epigastrium, by the general prevalence of nervous symptoms; by the affection being aggravated when the nervous symptoms undergo an exacerbation, and by the pulse and the action of the heart being natural during the intervals between the attacks.

To this category some would add, an increase of the palpitation after meals, or when the stomach is deranged, and amelioration produced by dyspeptic remedies; but, as the stomach produces the same effects when there is disease of the heart, these signs are not pathognomonic of nervous palpitation. To this point I would particularly direct the attention of practitioners; because many, in forming their diagnosis of the affections in question, regard the dyspeptic signs as paramount in value to all others.

Though nervous palpitation is often attended with various familiar nervous affections of the head, as pain or sensations of heat or of cold confined to particular parts and coming and going suddenly, temporary vertigo, tinnitus, and confusion of the sight, not increased by lying or stooping; it is not, when purely nervous, accompanied with genuine signs of cerebral determination or congestion: there is no universal throbbing headache with weight and tension, increased by stooping or the recumbent position: no stunning sounds and pains in the head on suddenly lying down or rising up: no permanent somnolency, apoplectic stupor, or regular apoplectic fits, as in hypertrophy, &c.

Physical signs.—" In nervous palpitation, says Laennec, the first impression which the application of the stethoscope to the region of the heart produces on the ear, shows at once that this organ has not great dimensions. The sound, although clear, is not loud over a great extent; and the shock even when it at first appears strong has little real impulsive force, for it does not sensibly elevate the head of the observer. This last sign appears to me the most important and the most certain of all when we add to it the frequency of the pulsations, which is always greater than in the natural state. Most commonly it is from eighty-four to ninety-six per minute." I transcribe this passage verbally, because it is a description of what I have so often designated the jerking impulse.

To these signs may in many instances be added intermittent bellows and sawing murmur of the heart and sometimes of the arteries, which I have elsewhere attributed to the morbid velocity with which the blood is propelled by the sudden and as it were spasmodic contraction of the heart. (Vid. p. 76.) The murmur occurs whenever the action of the organ is excited, and in some instances the slightest causes suffice to produce this effect, as a momentary mental emotion; a change of posture, from the recumbent to the erect, for instance; a constrained position; a meal; flatus in the stomach, &c. I have often found the phenomenon to subsist for a few seconds or minutes only, that is, so long as the exciting cause continued in operation. The patient, if asked whether he is conscious of palpitation, invariably replies in the affirmative; yet the pulse may not be strong—it may even be small and weak; but it will be "sharp or jerking." It is the velocity, therefore, not the power of the heart's contraction which causes the murmur.

The following cases are added, as exemplifying more graphically than can be done by a mere description, the manner in which nervous and organic affections of the heart run into each other, and the signs by which they may be distinguished.

CASE I.

A medical gentleman was subject for four years to dyspeptic symptoms and palpitation of the heart. They commenced about six months after leaving school where, while growing rapidly, he had been accustomed to very violent exercises and exertions. When he became an apprentice, his appetite was very great. He eat large quantities of animal food and never took any kind of exercise. His first symptoms were a heavy pulsating pain in the back of the head, extending forwards to the forehead, aggravated by any sudden motion, particularly that of rising up or lying down; giddiness, and disinclination to any exertion, sleep constantly disturbed by frightful dreams, particularly if the head was not much higher than the body; shortness of breath and palpitation on going up stairs or up a hill; a weak fluttering pulse when agitated or startled, accompanied by a sensation of weight and fulness about the heart. These symptoms continued for about three months when he had a fit of congestion of the head for which he was bled, blistered and purged. He now fell greatly into the habit of rowing, and made long and violent exertions in matches &c. but he was always short-winded during the effort and after it suffered from palpitation. At this time he entirely lost his appetite; his fingers were blue and very generally dead, particularly in the mornings, and his pulse feeble. He was seldom without heart-burn after any meal, and when this was the case he suffered more from palpitations, dreams, &c. and his fingers were more constantly dead. His feet also were nearly always dead, sometimes through the whole night. His bowels were irregular and costive, and skin cold and damp. By being bled occasionally when the affection of the head became worse, by abstaining from all violent exercise, paying attention to diet, correcting dyspepsia by occasional stomachics and antacids, and keeping the bowels open, he completely recovered from the above symptoms within four years from their first appearance, not having employed any systematic medical treatment for the first two. In the course of a year and a half after the commencement, he had few symptoms remaining, and he now considers himself quite well, never having any return of palpitation but when he has heart-burn, or has been taking too violent exercise.

This was a case of hypertrophy with a predominant degree of dilatation, as indicated by auscultation and percussion. Nervous affections of the heart never present the marked symptoms of cerebral determination exhibited in the present instance.

Case II.

Dyspepsia with Dilatation.

A Gentleman has from his youth been very subject to febrile and inflammatory affections and, though active, he has always been rather short-winded on ascending. He is subject to starting from sleep in consequence of which he awakes shouting out violently and always finds himself in a fit of palpitation and great agitation. When merely awakened from sleep, he always starts suddenly and in the same state of agitation. For at least ten or fifteen years he has always experienced great confusion in the head on first lying down to sleep: so much so, that he has seldom ventured to retire to bed alone. The same uneasy sensations have occurred on awakening in the morning and he has always required a quarter of an hour to compose himself before rising. Hence, he has always had a dread both of going to bed and of getting up. He has about eight times had a kind of fit, i. e. an indescribably distressing universal sensation, as if he were dying, invariably accompanied with palpitation and gasping and terminating in partial unconsciousness though without any convulsive movements. After an attack of this kind he immediately recovers the perfect command of his faculties. He has never had any paralytic sensations, though he is subject to tingling of the fingers, to a tremulous sensation of the left side and extremities and to sleeping of the feet. He has occasionally experienced optical illusions and once lost his sight completely, as if a blanket had fallen before his eyes.

He is subject to a throbbing pain in the posterior part of the head, which is one of his most distressing symptoms, and to acidity and excessive flatulence. A load of undigested and acid food is more apt than any other cause to bring on the fit described, and it is immediately relieved by an emetic, and sometimes even by a large dose of soda. Bowels regular, but evacuations generally unnatural.

Until three years ago he was in the habit of being cupped on the nape of the neck every three or four months and experienced great relief from the depletion. Since that time it has been less necessary and has only been resorted to occasionally.

The patient gets rid of nearly all the inconvenient symptoms when he pays strict attention to diet and regimen and relieves the circulation by cupping when the cerebral symptoms indicate it.

This is a case in which the symptoms were so closely connected with stomachic derangement that they were long supposed to result from the latter cause exclusively. The nature of the cerebral symptoms, however, and the evidence of dilatation afforded by auscultation, remove all doubt as to the real nature of the complaint.

A gentleman rather dyspeptic from his youth became affected with permanent depression of spirits from a mental cause. This was followed by excessive torpor of all the functions both corporeal and mental. Dyspepsia manifested itself in its most aggravated form, while the mind, naturally energetic and powerful, became obtuse and totally incapable of exertion and the spirits sunk into a state of apathetic despondency. With these symptoms he experienced palpitation on the slightest exertion or emotion. It sometimes consisted in merely a few rolling or tumbling movements of the heart attended with a sensation of fulness and oppression; at other times the organ fluttered and faltered for several minutes or for a longer period, the pulse being small and feeble and exhibiting the same unsteadiness; at other times again, the palpitation amounted to a violent paroxysm accompanied with gasping and orthopnœa. During the intervals of the attacks, he was neither short-winded nor subject to palpitation, and he invariably improved by exercise, which he is capable of taking to a great degree.

He was subject to occasional temporary local pains in the head with stupor, somnolency and sometimes with visual illusions.

This patient after suffering for upwards of four years, has almost completely recovered by a removal of the mental depression, by travelling as a pedestrian, by an abstemious dyspeptic diet, by strict attention to maintain regularity of the bowels, by combatting fits of dyspepsia immediately on their appearance, (for which evacuation of the stomach by an emetic, followed by abstinence and an aperient were the most efficient remedies,) and by pills consisting each of a grain of sulphate of iron, one of aloes, and three of comp. cinnamon powder, taken to the extent of one or two whenever the bowels were torpid, a lavement being employed when the pills failed.

In this case the sounds and action of the heart were natural except during the attacks of palpitation. The cerebral symptoms were partly nervous and partly those of a languid circulation through the head, but not of increased determination to it.

CHAPTER III.

SYNCOPE.

Causes of Syncope, 499. *Phenomena of*, 500. *Living Inhumation from*, 500. *Syncope in Asphysia, how the Persistance of Life may be ascertained*, 500. *Nervous Syncope not dangerous*, 500. *When from Disease of the Heart, especially with Angina Pectoris, dangerous*, 500. *Treatment of Nervous Syncope*, 501. *Of Organic*, 501.

Opposed to the state of over-excitement of the nerves of the heart, which we have been considering in the last chapters, is that of deficient excitement, the extreme degree of which constitutes *syncope*. Numerous agents have the effect of reducing, and even completely suspending, the contractile power of the heart. Such, for instance, are the depressing passions, certain scents, pain, violent shocks of the nervous system from accidents, sudden loss of blood, congestion of the heart from obstacles to the circulation; stupifying poisons, as hydro-cyanic acid, digitalis; certain miasms, as the plague, Indian cholera, pestilential fever; any agents, in short, which can, directly or indirectly, suspend for the moment the excitability of the heart.

The phenomena of syncope are too well known to require description: it may be said summarily that they are those of sudden death, except that in most cases, though not in all, the patient can be restored to life. The most ordinary duration of syncope is from a few seconds to a few minutes; but in certain rare cases it lasts for hours and even days, sometimes imitating death so perfectly as to lead to the horror-striking accident of living inhumation. In ordinary cases, the unconsciousness is seldom complete, and, though the pulse be imperceptible, feeble sounds of the heart's action may in general be distinctly heard. The latter is sometimes the case in individuals, who, after immersion in water or other causes of asphyxia, exhibit no apparent signs of life. Under these circumstances, therefore, auscultation should invariably be employed; for, so long as the sounds are heard, the patient is perfectly within the possibility of recovery.

Syncope, though free from danger when purely nervous, is a formidable accident when accompanying organic disease of the heart, as it is apt to terminate in sudden death, being, in fact, less the cause, than the symptom of a fatal suspension of the circulation. This catastrophe is more liable to occur when angina pectoris is superadded to organic disease; in consequence, apparently, of the lesion being double, the motive principle as well as the muscular apparatus of the heart, being inadequate to the discharge of its function.

Treatment.—The ordinary excitants, which suffice for so slight an affection as purely nervous syncope, are, fresh air, the sudden aspersion of cold water, startling the patient by a sudden noise or blow, ammonia and other pungent errhines. When syncope is symptomatic of another disease, it requires that, in addition to its ordinary treatment, remedies should be employed adapted to the nature of the primary affection. The principles according to which this must be done in reference to disease of the heart and angina pectoris, have been explained in the preceding pages.

PART V.

MISCELLANEOUS AFFECTIONS.

THESE affections consist of a few which are not reducible to any of the preceding heads.

CHAPTER I.

POLYPUS OF THE HEART.

Discrepant Opinions respecting Polypus, 502. *Some are formed before and some after Death,* 503. Anatomical Characters, 504. *Of Polypi formed during or after Dissolution,* 504. *Of those formed previous,* 504. *Of those formed long previous,* 505. *Of globular Vegetations of Laennec,* 506. *Two Opinions respecting the* Formation of Polypi, 507. 1st. *by Inflammation,* 507. *This Opinion inaccurate,* 507. 2d. *by Stagnation of the Blood,* 508. *How Polypi become adherent explained,* 509. *Polypi are possessed of Vitality and susceptible of Organization,* 509. Signs and Diagnosis *of Polypi,* 509. *Signs of Laennec,* 510. *Signs of the Writer,* 511.

THE concretions of blood commonly called polypi of the heart and great vessels have given rise to much discussion, and various doctrines respecting them have successively superseded each other in the schools. According to some, they are merely coagula of blood formed after death: according to others, they are organized substances, formed before death, and analagous to nasal and uterine polypi: others, again, believing that both kinds existed, denominated the former *false* and the latter *true*.

It was a very general opinion during the last century, that polypi produced all the symptoms which are now known to result from organic disease of the heart: while some, on the contrary, doubted whether they produced any symptoms whatever. The researches of Corvisart, Testa, Burns, Kreysig, Laennec and succeeding pathologists have decided the question, and have fully established the fact, that there are some polypi formed after, or during the period of dissolution, and others formed for a longer or shorter period anterior to it, and the cause of certain well marked symptoms during life.

That the latter species should exist, might be anticipated a priori from the fact that, in the arteries and veins, blood can coagulate during life and, becoming organized and adherent to the walls, obliterate the canal of the vessel. Instances of this have, of late years, been accumulated in abundance by Hodgson, Burns, Kreysig, Bertin and Bouillaud, Laennec, Velpeau and others: in short, there is scarcely a single considerable vessel, especially a vein, in which the concretions in question have not been found. In veins, they are often the cause of partial dropsies.*

Anatomical characters of polypi of the heart.—Polypi formed after death or during the last moments of life, are concretions of fibrine, which, if very recent, merely overspread portions, but seldom the whole, of the clots of blood in the heart and great vessels with a thin, translucent layer resembling inflammatory buff: but, if rather older, they constitute larger and thicker masses, often entirely independent of the clots of blood. In dropsical subjects or when the blood is very serous, the fibrine appears as it were infiltrated, and is soft, trembling, and semi-transparent like jelly. The polypi of the above kinds are more common on the right side of the heart than the left; they do not adhere to the walls; their fibrous texture is very indistinct, and they are of a uniform semi-transparent yellowish or whitish colour: by these characters they may be distinguished from polypi formed some time previous to death.

The latter are of a much firmer consistence; more opake, and less charged with serum; their fibrous texture is more distinct; they are often arranged in concentric layers; their colour, instead of being uniformly whitish or yellowish, has in parts a pale flesh tint sometimes slightly violet; they are found more frequently on the left side of the heart than recent polypi are; and they adhere more or less firmly to the walls of the heart, from which it is scarcely possible to draw them away in a single piece, as the extremities remain attached under the columnæ carneæ. The medium of adhesion is a filamentous tissue, the rupture of which leaves a roughness both on the lining membrane of the heart and on the surface of the polypus. The surface also presents spots of blood penetrating more or less deeply and sometimes ramifying inwards, as if to form vessels for the purpose of organizing the mass—for there is no doubt that polypi do become more or less organized, and there is every reason to believe that the process is the same as in false membranes.

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* Vid. M. Bouillaud Archiv. gen. de Med. tom. ii. et v.

There are other polypi which appear to be of still older formation, and which may probably be dated as far back as several months prior to the death of the patient. They are completely opake like paste or cheese, exactly resemble the oldest fibrinous layers of false aneurisms, and adhere so firmly to the walls of the heart that they cannot be detached without scraping with the scalpel and sometimes without removing the internal membrane.

To the class of polypi are also in my opinion, to be referred the *globular vegetations* of M. Laennec.[*] They present themselves under the form of irregularly spherical or ovoid balls or cysts, the size of which varies from that of a pea to that of a pigeon's egg. The cysts are smooth externally; and their walls, which scarcely exceed half a line in thickness, are composed of an organized substance somewhat firmer than the white of a hard-boiled egg, and resembling in opacity the oldest polypous concretions. The internal surface of the cyst is less smooth than its exterior, and appears formed of a softer substance, which sometimes gradually degenerates, in the direction from without to within, into a matter similar to the contents of the cyst. These contents, in the cysts which there is reason to believe the most recent, are bloody; in the older they are like lees of wine, and in the oldest they are puriform. The cyst adheres by a pedicle, which, according to M. Laennec, is of more recent formation than the cyst itself, being more translucent and in a less advanced state of organization. The pedicle is interlaced amongst the columnæ carneæ and united more or less firmly with the internal membrane. The most common situation for these bodies, and where I have frequently found them, is about the apex of the ventricles. I am not aware that they are ever found in the great vessels: I have never seen them there.

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[*] De l'Auscult. tom. ii. p. 630.

Formation of polypous concretions.—Two opinions have been entertained respecting the formation of polypi: 1. some have attributed them to inflammation: 2. others, to retardation of the blood.

1. The advocates of the former opinion rest on the fact that there is sometimes an intimate adherence or continuity of substance between the polypus and the internal membrane of the heart and vessels. Now, it cannot be denied that sometimes, especially in the veins where the circulation is slow, an inflammation of their internal surface occasioning the formation of a false membrane, may be the cause of a concretion of blood, which, being imbibed by the false membrane, swells it, and forms a coagulum around it, as if by a sort of attraction. Yet there is a conclusive argument to prove that this is not the process by which polypi are formed in the heart: viz. were they so formed, they would, from the first, be adherent; which is not the case: the comparison of facts clearly proves

that, in the first instance, they are free, and that it is only the older and more perfectly organized which become adherent.

Again, to imagine that inflammation of the internal surface of the heart acts upon, and coagulates the blood, is not only a wholly unsupported hypothesis, but is directly opposed to facts; for it is not in youthful subjects, in the plethoric, in those full of life and eminently disposed to the inflammatory orgasm, that polypi form in the heart: but, on the contrary, they form during the last moments of life in all diseases, and especially of chronic disease, which have occasioned cachexy, marasmus, extreme debility, and which have been accompanied by a local or general obstacle to the circulation. Hence it is evident that inflammation is not the cause of polypi.

2. According to the second opinion they are attributable to retardation and consequent stagnation of the blood; which to me appears by far the more rational view. It has just been shown that they occur in the circumstances most favourable to stagnation. That it alone, independent of any action of the containing organs upon the blood, is capable of causing the separation of the fibrine from the other parts, is proved by this separation taking place in blood drawn by the lancet, and in men and animals that have died a violent death

during a state of perfect health. We have, further, an exemplification of the whole process of stagnation and coagulation in false aneurisms.

The adhesion of a polypus appears to be occasioned by the irritating action of the body itself on the walls of the heart; whence there results an exudation of lymph on the latter, which forms the agglutinating medium. This process is very manifest in the veins; for, in them, recent coagula are not adherent; and a firm false membrane, strongly united to the walls of the vein, is never found but in the points where the firmness and partial desiccation of the coagulum, its consisting of fibrine changed in various gradations, and sometimes the contraction of the vein, show that the concretion is old: whence it is to be inferred that the false membrane was a result of inflammation occasioned by the irritation of the concretion.

It is demonstrated by observation that fibrine, separated from the blood and become concrete in a living organ, (whether the heart, or a serous or other membrane into which it had been extravasated) retains its vitality and is susceptible of organization in an equal degree with inflammatory lymph. This may be an additional reason to account for the facility with which polypi form adhesions.

Signs and diagnosis of polypi of the heart.—I progress of the disease, or if they are such as the disease could not be supposed capable of producing, there is strong reason to suspect a polypus.

The small *globular* polypi often exist without producing any obstacle to the circulation, or any irregularity of the action of the heart. In general, however, they are found in those who have been in a moribund state for many days and sometimes many weeks before death.

One of the greatest dangers of excessive blood-letting or otherwise reducing the system, and of the indiscreet exhibition of nauseants and digitalis, in advanced stages of organic disease of the heart, arises from the risk of the formation of polypi in consequence of languor of the circulation.

CHAPTER II.

DISPLACEMENTS OF THE HEART.

Causes of Displacement, 513. *Symptoms*, 513. *Means of Diagnosis*, 513.

The heart being sustained in its place principally by the equal pressure of the lungs on all sides, may be displaced when that pressure is rendered unequal. I have seen this occur from pneumothorax, by which the organ was forced completely to the right of the sternum; by the same affection with hydro-thorax producing a similar effect; by hydro-thorax alone, (case of Rowe) by aneurism of the ascending aorta, displacing it to the left (case of Hill) by enlargement of the liver; and by an enormous fungus hæmatodes of the right lung. It may also be displaced by emphysema of the lungs; by tumors in the anterior mediastinum, and by aneurisms of the arch of the aorta. The two latter causes generally force it downwards. When the heart is enlarged, it pulsates lower in the chest than natural, and this is an excellent sign of enlargement.

Slight displacements occasion little inconvenience: when considerable, they may create serious functional derangement, especially palpitation.

Displacements are easily detected by auscultation and percussion.

CHAPTER III.

HYDRO-PERICARDIUM.

When an Exudation during or after Dissolution, 514. *The Quantity in Cases of general Dropsy*, 514. *General Signs obscure*, 514. *No physical Signs given by Laennec*, 515. *Physical Signs by the Writer*, 515. *Treatment*, 516. *Tapping*, 516.

SEROUS effusion in the pericardium is extremely common as an attendant of general dropsy, but very rare as an idiopathic disease. When it does not exceed three or four ounces in cases in which the serous diathesis prevails, it may be merely an exudation which has taken place during the last moments of life; and when it does not exceed one or two ounces in ordinary cases, it may be ascribed to the same cause.

In general dropsy, the pericardium usually contains less fluid, in proportion, than other serous cavities. The quantity seldom exceeds one or two pints, but Corvisart has once seen it amount to eight. The fluid is sometimes colourless, but usually it is yellowish or brownish, though transparent and free from albuminous flakes: occasionally, though very rarely, it is bloody.

The signs of hydro-pericardium are obscure.

The weight in the region of the heart, the sensation of the organ floating, experienced by the patient, undulations of the fluid felt and even seen in the intervals between the third, fourth, and fifth ribs, irregular action of the heart, a small, frequent and intermittent pulse, orthopnœa, palpitation and syncope, are signs common to other complaints and therefore unworthy of confidence, except as corroborating others more characteristic.

Laennec expresses himself unable to say what signs auscultation will supply, but thinks that effusions less than a pint will not afford any; and that we shall probably never be able to detect hydropericardium which is not even much more considerable. After much attention to this subject, I think it is in general possible to detect from eight or ten ounces upwards* by the following signs.

Dulness on percussion is preternaturally extensive; the motions of the heart are perceptible beyond the ordinary limits; the impulse is of an undulatory nature, some beats being stronger than others, and the point at which they are most sensible, varying every moment; the impulse does not accurately coincide with the sound of the ventricular contraction, as the heart has to remove the interposed fluid before it can impinge against the thoracic walls; finally, the sensation communicated to the hand and the stethoscope is that of an impulse transmitted through a fluid, and not of an organ striking the ribs *immediately*.

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* In the cases of Bryant and Snowden a much less quantity was detected, but I would not venture to say that so little could always be recognised.

Hydro-pericardium from general dropsy requires the same treatment as the dropsy. For idiopathic hydro-pericardium, since other remedies are almost unavailing, tapping has been suggested by Senac, countenanced by Laennec, and practised, but unsuccessfully, by Desault and others. To myself the operation appears inadmissible; for, independent of its danger, unless adhesion were effected by exciting pericarditis, the fluid would probably be regenerated, as in hydrocele and ascites. Laennec thinks that the least dangerous mode of operation would be, that of trepanning the sternum above the xiphoid cartilage, as thus the pleura would not be opened, and the diagnosis might be verified by inspection before the pericardium was punctured.

CHAPTER IV.

PNEUMO-PERICARDIUM.

Generally from Putrefaction, 517. *Sometimes probably anterior to Death*, 517. *Conjoined with liquid Effusion*, 517. *Signs*, 517. *Laennec's Opinion that it is present in almost all Cases in which the Beats of the Heart are audible at some Distance from the Chest*, 517. *Case of great Pneumo-pericardium*, 518.

LAENNEC assigns this name to effusions of air within the pericardium, which are very frequently found on dissection. In subjects that have been kept for some time, the effusion is to be ascribed to decomposition; but in many others, judging from the absence of all signs of putrefaction, it is anterior to death. Most frequently it is conjoined with a liquid effusion, and the two may take place simultaneously in the last moments of life in any disease. Laennec states that he has sometimes detected it by an unusually clear resonance at the base of the sternum which had supervened within a few days, or by a sound of fluctuation attending the beats of the heart and strong inspirations. Though he has not had an opportunity of verifying the fact, he is convinced that in almost all the cases in which the beats of the heart could be heard at a certain distance from the chest, this phenomenon is due to the temporary effusion of a gas, which is in general promptly absorbed, and the presence of which in the pericardium creates no serious inconvenience.* This is very questionable.

Air is sometimes found on dissection in the cavities of the heart. Dr. Forbes of Chichester recently favoured me with the following communication, "I yesterday examined a boy who had died suddenly after being affected for years with all the symptoms of extreme dilatation of the heart. I found the organ very large from dilation of both ventricles, and both were distended with air—in all eight or ten ounces. There was no particular putridity, the boy having been dead only thirty-six hours." A similar case is recorded in Simmons's Lond. Med. Journal. part 3rd, for 1785. As air in the ventricles is incompatible with the maintenance of life, it must, in these cases, have been generated, or conveyed there, after death; but if putrefaction be its cause, it is remarkable that the phenomenon is so rare.

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* De l'Auscult. t. ii. p. 672 and 455.

PART VI.

CASES.

THE following cases, collected at St. George's Hospital, though few in number, present well characterized exemplifications of, I believe, all the ordinary, and many of the more rare diseases of the heart: also of the general histories and signs given in the antecedent parts of the work. I have, for the sake of brevity, omitted the physical signs of pulmonary affections, but have adverted to the affections in the diagnosis and succinctly described them in the post-mortem examinations.

I have likewise omitted details of treatment; for, though pre-eminently important to the observer, they afford comparatively little instruction to the mere reader, by whom, in consequence, they are seldom perused. The first prescriptions I have introduced; for, coming from the pens of physicians not only eminent in their profession, but remarkably conversant with the class of diseases under consideration, they form a valuable therapeutic code. Having found it impossible perfectly to classify the cases under the heads of hypertrophy, dilatation, valvular disease, &c. in consequence of these affections being in general complicated with each other, I have merely thrown the several classes rudely together, as far as practicable, and have appended an Analytic Table which will afford every facility of reference.*

Case I.

Great hypertrophy with dilatation; hydro-pericardium; emphysema; peripneumony.

Robert Bryant, æt. forty-two, of sallow and livid complexion, was admitted into St. George's Hospital under Dr. Chambers, May 6, 1829, with œdema of the lower extremities, cough, dyspnœa

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[*] Minutes of the physical signs of disease of the heart were taken by other gentlemen in the hospital as well as myself: more especially by Mr. Johnson, at present House Surgeon to the Institution;—a gentleman no less remarkable for an intimate knowledge of auscultation, than for general talent combined with a sound judgment and a liberal mind. I have pleasure in stating in corroboration of the accuracy of my own minutes, that those of Mr. Johnson coincided with them closely and often verbally, though we had no communication until both were written.

As I have endeavoured to explain in the Lond. Med. Gaz. August 21st and 28th, and December 25th, 1830, how the physical signs presented by the following cases are irreconcileable with any view of the heart's action but that which I have proposed, it is unnecessary to repeat the explanation in this volume.

and palpitation increased on exertion, starting from sleep, great pulsation of the jugular veins, especially the right, pulse 100 full and strong, urine free but thick.

Had dropsy thirteen years ago. The present symptoms came on three months ago, commencing with cough. Was previously in good general health, and did not complain of shortness of breath.

The *resonance* of the præcordial region is dull over an expanse of five inches in diameter. The *impulse* of the left ventricle is strong, extensive and undulating, with a violent receding jerk or shock when the heart retires. The first *sound* of the left ventricle is scarcely audible, but the second sound is sufficiently smart and loud.

Diagnosis.—Hypertrophy and dilatation of the heart. Hydro-pericardium. Little if any hydrothorax. Lungs gorged and emphysematous.

℞ Pil. Hydr. gr. v. Scillæ pulv. gr. i. Pil. bis die s. ℞ Haust. nitri, Sp. æth. nitric.—Junip. C. $\overline{\text{aa}}$ ʒi m. f^{t}. haust ter die. ℞ Haust. sennæ, Tr. Jalapæ ʒi m. f^{t}. haust o. m. Diæta lactea.

Five days after admission he was attacked with peripneumony, for which ℥xii of blood were drawn, and ℥x four days afterward. Sputa viscous and rust-coloured, pulse became irregular, sleep disturbed. (Cont. Med.) Was relieved for two or three days, when he had a violent attack of palpitation and orthopnœa. Pulse 110, sputa bloody, mucous râles in the throat and lungs (V. S. anodynes and diaphoretics). The paroxysm subsided in 36 hours, but he gradually sunk, and died on the 23rd.

Autopsy.—Left ventricle immensely hypertrophous; right, considerably: both, dilated: valves sound: ℥iv of serum in the pericardium: ℥ij in the cavities of the chest. *Lungs.* Hepatization of the inferior lobes on both sides. It is sero-purulent, of chocolate colour and very flaccid and lacerable. Parts of the middle lobes are in the first degree of peripneumony. The remainder of both lungs is bloated with emphysema and œdema.

Remarks.—The hypertrophy occasioned the power of the impulse, the dulness of the first sound, and the strength of the pulse; while the dilatation rendered the second sound sufficiently loud and smart, the pulse full, and the impulse and præcordial non-resonance more extensive than in hypertrophy alone. The great predominance of the hypertrophy over the dilatation prevented the latter from increasing the first sound. The violence of the back-stroke resulted from the hypertrophy and dilatation conjointly; as the augmented weight of the heart, and the increased influx of blood during the ventricular diastole, conspired to render that motion boisterous. The fluid in the pericardium imparted the undulating character to the impulse. This is in consequence of the fluid being displaced by the motions of the organ; and as these motions are more violent in cases of hypertrophy, the undulation is proportionably greater. The pulse, at first regular, as is generally the case in uncomplicated hypertrophy, became irregular in consequence of the engorgement of the heart occasioned by the pulmonary obstruction and the reduction of the vital powers.

As it is almost certain that the hypertrophy existed to a greater or less degree, at the time of his former dropsical attack thirteen years before, and as he had remained during the interval without complaining of bad health, the case tends to show that hypertrophy, in its simple state, is not attended with much inconvenience.

Emphysema is one of the most dangerous complications of peripneumony; for, as the dyspnœa which it occasions is liable to be attributed solely to the inflammation, blood-letting may be carried to excess; and thus, the vital powers being diminished while the obstruction in the lungs remains, the patient sinks suddenly and unexpectedly. Several cases of this description have fallen under my observation. The present is not of that number, as the emphysema was detected by Dr. Chambers, and the depletion judiciously regulated accordingly.

CASE II.

Hypertrophy of the left ventricle, with cartilaginous contraction of the aortic valves and ossification and contraction of the aorta.

Mary Andrews, æt. 40, was admitted into St. George's Hospital, under Dr. Seymour, May 9th, 1829, in an asthmatic paroxysm, which rendered her incapable of giving any account of herself. Violent action of the heart, countenance anxious, pulse full, strong, and rather behind the ventricular systole.

Auscultation.—Impulse increased in force and extent; felt in the epigastrium. Left ventricular sound is attended with loud and prolonged rasping murmur.

Diagnosis.—Organic disease of the heart; contraction of an orifice; ossification.

Was bled to ℥x, and antispasmodic and sedative draughts. Died in the evening.

Autopsy.—Simple hypertrophy of the left ventricle; the free margins of the aortic valves are stiff with cartilage, which prevents them from closing perfectly. The interior of the aorta beyond the valves is ossified, scaly, rigid and contracted.

Remarks.—The hypertrophy was the cause of the increased impulse, which exceeded what would be occasioned by mere palpitation: also of the full, strong pulse. Contraction of an orifice was indicated by the rasping-murmur; and that the aorta was its seat, was almost certain from the hypertrophy of the ventricle, (this being generally occasioned by an obstruction in front) and from the fullness, strength and regularity of the pulse, which characters it would not have possessed had there been regurgitation through the mitral valve. It is remarkable that such a degree of valvular obstruction as existed in the present case, produces no sensible effect on the pulse. Even a considerable degree does not diminish its strength and tension, though it reduces its size; and it is only an extreme degree which renders it small, weak and irregular.

The rasping-murmur was attributable to the osseous asperity of the aorta; as the smooth cartilaginous disease of the sigmoids was only calculated to produce the softer sound of bellows. There can be little doubt that contraction and ruggedness of the aorta, by impeding the circulation, occasioned the hypertrophy of the left ventricle; and that by checking the current of the blood, it rendered the radial pulse later than the ventricular systole.

Case III.

Enormous dilatation with hypertrophy of both ventricles; enlarged liver. Fits from cerebral congestion.

Richard Collard, æt. 36, a coach-maker, of large frame, but emaciated and affected with jaundice, was admitted into St. George's Hospital, under Dr. Chambers, August 19th, 1829, with ascites; great œdema of the legs; dyspnœa, exasperated by every movement; cough; great pulsation of the carotids; varicose intumescence and undulation of the jugulars; impulse of the heart preternaturally strong and extensive; pulse bounding but not hard, moderately full and rather vibrating; skin clammy; tongue whitish; bowels open; evacuations of a light clay-colour; urine scanty and deep-coloured. Liver is felt to be enlarged.

Has been more or less ill for two years. Complaint is attributed to fretting. It commenced with shortness of breath and loss of appetite. Dropsy first appeared six months ago, and skin became yellow five or six weeks ago. Is said to be subject to fits.

Auscultation.—Resonance very dull over the whole of the præcordial region. *Impulse* is a powerful heaving, terminating in a jerk or back-stroke: it is felt much more extensively than natural, and in the epigastrium. Both *sounds* are louder, and the first a little more brief, than natural. Above the clavicles there is a slight impulse with very feeble purring tremor and a whizzing sound, not loud or hoarse.

Diagnosis.—Great hypertrophy with dilatation of the heart; enlargement of the liver.

℞ ung. hydr. fort. ʒi hepatis regioni om. noct. infricand—Haust nitri, sp. æth. nitrici ʒi m. f^{t}. haust. ter die—Potassæ supertart ℥ss, jalapæ pulv. gr. x, om. noct. sumend.

During the first week he had three fits, which consisted of stupor, with slight convulsions and stertor, succeeded by sleep. The last attack was of two hours duration. The dropsy was greatly reduced by the remedies; but he sank exhausted on the eighteenth day after admission.

Autopsy.—Heart double its natural size, and, as he was of large frame, it was enormous. Left ventricle would contain a full-sized lemon, and the parietes were three-fourths of an inch thick. Right ventricle was similarly affected, but in a rather less degree. The muscular substance was pale and somewhat softened: it presented a mottled appearance. *Valves* and aorta natural. *Hydrothorax* to four pints. *Lungs* gorged with serum. *Mucous membrane* of the bronchi vascular and of a dim red colour. *Liver* twice its natural size, of intense yellow (ochre) colour, and its acini were enlarged in every degree up to the size of a pea. *Brain* healthy, but fluid under the arochnoid membrane.

Remarks.—The extraordinary degree of hypertrophy with dilatation was distinctly marked by the extensive dulness of the præcordial region on percussion, without signs of hydro-pericardium; by the powerful heaving and back-stroke; and by the loudness of both sounds. The predominance of the dilatation over the hypertrophy, prevented the pulse from being so hard and incompressible as the hypertrophy would otherwise have rendered it. Pulsation, vibration and whizzing sound of the larger arteries, as in the present case, are not uncommon phænomena when an increased quantity of blood is transmitted through the vessels with augmented force. They may easily be distinguished from the same resulting from aortic dilatation or disease, by the superior hoarseness of the sound and vigour of the impulse in the latter affections. It is probable that the hypertrophy originated in nervous palpitation, excited by the mental inquietude. The disease of the liver was most likely a result of congestion occasioned by impeded circulation of blood through the heart and lungs. The varicose and tumid state of the jugular veins depended on the same cause, while their pulsation was due to the hypertrophy of the right ventricle. The fits were dependent on the violent determination of blood to the brain. I have in many instances known such attacks recur repeatedly and at last terminate in a fatal apoplectic seizure,—a common result of hypertrophy of the left ventricle. Individuals reduced by years or disease often sink suddenly, as in the present instance, after the disappearance of much dropsical infiltration. The older physicians supposed that this was in consequence of accumulations in the great cavities; but as, in a large proportion of cases, dissection disproves the existence of such accumulations, dissolution must be ascribed to a failure of the vital powers.

Case IV.

Hypertrophy of the left ventricle: dilatation of both; disease of the interior of the aorta: angina; emphysema and œdema of the lungs.

David Keith, æt. 70, emaciated, of middle stature, and sallow complexion, with a circumscribed red of the cheeks, was received into St. George's Hospital, under Dr. Seymour, September 2d, 1829, subject to severe pain at the inferior part of the sternum and across the epigastrium, which comes on about midnight accompanied with orthopnœa amounting almost to suffocation. The paroxysm lasts several hours. Cough; dyspnœa on motion; cannot expand either side of the chest; its resonance is good, and in some parts (viz. the anterior and superior) it is more sonorous than natural; pulse 116, large and strong; bowels regular; flatulence.

Says that the asthmatic fits commenced only two months ago; and he attributes them to a "violent cough" which had existed for two months previously. Anterior to that time he did not suffer from dyspnœa. Has taken aperients and been bled.

Auscultation when the circulation was tranquil. *Impulse* of the heart not considerable. Both *sounds* are short and flat, and so loud as to be distinctly audible above the right clavicle. They are obscured below by the catarrhal râles.

Diagnosis.—Dilatation of the heart. Emphysema of the lungs; ædema of the lower lobes; chronic bronchial catarrh.

℞ sp. æth. sulph. ʒss, mist. camph. ʒx, bis die—℞ Hydr. submu. gr. ij, Pulv. Jacobi et Pil. sapon. cum opio āā gr. v. fiant pilulæ iij omni nocte sumendæ.

The asthmatic attacks were diminished for a week, but they recurred with aggravated violence in consequence of his taking fresh cold. October

13th, cough worse, with inability to expectorate from weakness; dyspnœa, emaciation and paleness are increased; voice a whisper; pulse 98 rather unequal; tongue dry; thirst; anorexia; loud sonorous râles over the whole anterior chest. These symptoms increased and he expired on the 28th October.

Autopsy.—Left ventricle was an inch thick and dilated to about one half larger than natural. Muscular fibre red and firm. Right ventricle dilated to the same extent, but not thickened. Margins of the valves in parts slightly thickened with fibro-cartilage, but not sufficiently to cause symptoms. *Aorta*, to the extent of an inch and a half above the valves, very slightly dilated, and opposite to the origin of the left subclavian somewhat contracted. Its interior universally overspread with firm, cheese-like matter, intermixed with a few calcareous scales. This state extended to the pelvic bifurcation.

Lungs.—extremely large, and did not callapse when the chest was opened, in consequence of being universally distended by emphysema and œdema. Air vesicles enlarged—some to the size of pin's heads, and their insufflated state rendered many of the superficial lobules prominent and perfectly pale. Spumous serum exuded copiously on pressure. The lower lobes were in the state of chocolate coloured flaccid hepatization, heavier than water, but not purulent or lacerable. Some of the great bronchi were of an indelible brownish red colour and contained purulent mucus.

Remarks.—The interposition of the bloated lungs between the heart and the thoracic parietes prevented the resonance of the præcordial region from being so dull, and the impulse of the organ from being so strong as such a degree of hypertrophy and dilatation would otherwise have rendered them. The action of the heart was not proportionate in violence to the extent of the enlargement, owing, perhaps to the advanced age and great emaciation and exhaustion of the patient. The existence of murmur from the ruggedness of the aorta could not be ascertained, in consequence of the loudness of the pulmonary râles.

In this present, as in many similar cases, the disease of the heart was called into fatal activity by the superadded impediment to the circulation from emphysema and œdema, &c. of the lungs. The supervention of the asthmatic fit during the night was favoured by the recumbent position, and by the accumulation of mucus during sleep. The pain in the region of the heart, commonly called angina pectoris, must be referred to nervous irritation occasioned by the gorged and labouring state of the organ. This case is interesting and important as exemplifying the connection which generally subsists between angina and organic disease of the heart.

Case V.

Hypertrophy and dilatation after pericarditis; pneumonia.

John Green, æt. 43, a groom, of middle stature and pale, fair complexion, was received into St. George's Hospital under Dr. Chambers, January 6th, 1830, with "a weight in the chest;" dyspnœa and palpitation increased by any exertion; cough; viscid, rust-coloured sputa, sometimes black with grumous blood; hoarseness; orthopnœa; pulse 120 sharp; tongue thickly furred and yellow in the centre, pale at the edges; thirst; anorexia; bowels costive; urine scanty and offensive; emaciation.

Had been suddenly attacked, three months before, with dyspnœa, palpitation and the other symptoms. They had been occasionally relieved, but, on his admission, were worse than ever. Previous to the attack he was healthy.

Auscultation.—Increased sound and impulse of the heart.

Diagnosis.—Peripneumony; enlargement of the heart.—V. S. ad ℥xij. ℞ Haust. salin. cum oxym. scillæ ʒss. 6^{tis} horis. ℞ Hydr. submu. gr. v. hac of the præcordial region extremely dull ; pulse 120 full, strong and regular, but *compressible*—a circumstance particularly pointed out to me by Dr. Chambers.

He had suffered from acute rheumatism eight years before, and had never since been exempt from palpitation.

Auscultation was not employed.

Diagnosis.—Acute rheumatism ; organic disease of the heart ; adhesion of the pericardium.—℞ Pil. Hydr. gr. iij, pulv. scillæ gr. ij, pulv. digitalis gr. ss, ter die—℞ Inf. aurant. c. ʒx, Sp. æth. nit. et sp. junip. c. āā ʒi, T^r. hyoscyami m x, 6^{tis} horis. ℞ Haust. sennæ, pulv. Jalapæ gr. vi, potassæ supertart. ʒi, alterno die.

He died within twenty-four hours, after an attack of hæmoptysis to a considerable extent.

Autopsy.—Universal adhesion of the pericardium. The layer of lymph, forming the medium of adhesion, was thin and dense. The heart was judged to be nearly three times its natural dimensions. The enlargement was principally in the left ventricle, the walls of which were an inch and a half thick, and the cavity larger than the largest orange. The right ventricle was similarly affected but in a less degree. The *aortic valves* were thickened, nodulated and corrugated by an opake, yellow degeneration, partly cartilaginous and partly steatomatous. This had caused the detachment of the angular extremities of the valves from their insertions; so that, being adherent by their centres only, they projected loose into the artery, and were destitute of fulcra by which to oppose the reflux of blood from the aorta.

Remarks.—The degree of enlargement which existed in this case is seldom exceeded. There is little doubt that the affection originated in the attack of rheumatic pericarditis eight years before, by which adhesion was occasioned—a lesion that never fails to induce more or less dilatation and generally hypertrophy. As the dilatation was so enormous, it is not improbable that softening from the pericarditis contributed in the first instance to its production. The thinness and density of the lymph indicated the oldness of the adhesion; for in recent cases the deposition is always soft and usually several lines in thickness. The adhesion was inferred from the obvious hypertrophy with dilatation, the antecedence of rheumatic pericarditis, and the remarkable movement in the epigastrium, probably occasioned by retraction, as supposed by Dr. Sanders. This is one of the very few cases in which I have observed this phenomenon, nor can I say that the retraction was very distinct even here. Did regurgitation of the aortic blood consequent on the disabled state of the valves, occasion the compressibility of the pulse, noticed by Dr. Chambers? It is manifest that such regurgitation must have a powerful effect in producing enlargement of the left ventricle; for the whole weight of the arterial circulation, instead of being sustained partly by the valves, rests constantly, and exerts its expanding force, upon the ventricle.

The hæmoptysis depended on the state of the heart. For, as the retrograde pressure of blood in the left ventricle had precisely the same effect as a valvular obstruction in opposing the passage of blood from the lungs through the left side of the heart; while, at the same time, the right ventricle, hypertrophous and dilatated, expelled a preternatural quantity of blood with augmented impetuosity, the delicate vessels of the lungs, exposed to these conjoint forces operating in opposite directions, yielded to the pressure, and relieved themselves by transudation of blood into the air passages. It is for this obvious reason that pulmonary apoplexy and hæmoptysis are more frequently found connected with an impediment on the left side of the heart, and simultaneous hypertrophy of the right ventricle, than with any other lesions of the organ.

CASE VII.

Dilatation of the heart; hydro-pericardium; hydrothorax.

John Snowden, æt. 38, tall, thin, sallow, with circumscribed redness of the cheeks, was received into St. George's Hospital under Dr. Seymour, May 19th, 1829, with orthopnœa; excessive dyspnœa on the slightest exertion; cough; watery expectoration; ascites; face puffed and leucophlegmatic; great œdema of the legs; undulating or rolling motion in the præcordial region; pulse 110, weak and irregular; urine scanty.

He had long been short-winded, but to no considerable degree until two months before admission, when he was suddenly attacked with excessive dyspnœa while walking. This symptom increased, and in a fortnight was followed by dropsy.

Auscultation.—Resonance dull over an unusual extent of the præcordial region. *Impulse* of the left ventricle is slightly increased, but *undulating* and *not synchronous with the ventricular contraction* as indicated by the first sound. Over the right ventricle the impulse is weaker. *Sounds*, are short, flat, and audible over the whole anterior surface of the chest. Neither coincides with the radial pulse, and they are so much alike as to be with difficulty discriminated.

Diagnosis.—Dilatation of the left ventricle, without attenuation of its parietes; hydro-pericardium; hydro-thorax. (ascertained by percussion.)

V. S. ad ℥x—℞ Elaterii gr. i, calomel. gr. ij, cras mane.—Potûs supertart. potassæ ℔i in die. He improved considerably for a fortnight, when he was bled to ℥x for increase of cough and slight erysipelas of the face. These were mitigated, but the debility and dropsy increased. Pulse 120 (Haust. sulph. quinæ ter die.—℞ Haust. opiat., oxymel scillæ et sp. æth. nit. āā ʒss omni nocte.) In a fortnight the cough and dropsy were greatly diminished, and he was able to leave his bed a little.

Auscultation.—The impulse coincides better with the ventricular systole, and the heart is more distinctly felt to strike the ribs. Pulse is still weak, and not perfectly synchronous with the ventricular contraction. *Diag.—Hydro-pericardium diminished.* Emaciation and debility now made rapid strides, the dropsy began to re-accumulate and in another fortnight he sank.

Autopsy.—Upwards of Oij of serum in the pleura; ℥iii or iv of bloody fluid in the pericardium. *Lungs.* The left was healthy above, but the inferior lobe was gorged with blood, and heavier than water. The right contained some suppurating tubercles. *Heart.* Left ventricle was considerably dilated, and its parietes were half an inch thick. The right ventricle was rather less dilated than the left, and its parietes natural, or thinner if either. *Valves* healthy. The apex of the left ventricle contained a polypus which had softened or suppurated in its centre; and roundish nodules of lymph were found in the instertices of the columnæ carneæ.

Remarks.—The short, flat and loud first sound, and the weak and irregular pulse indicated the dilatation; while the rather increased action of the left ventricle, though partly attributable to the accelerated and disturbed state of the circulation, denoted that the parietes were not attenuated.

In cases of dilatation, when the general constitutional powers are still tolerably good, and the increased capacity of the heart does not greatly predominate over its muscular strength, the pulse is generally regular, and, though soft, it has frequently a considerable degree of fulness. But when the strength fails, as in the present instance, or the heart is otherwise excited beyond its contractile power, the same pulse may become both weak and irregular. The latter character, therefore, must be regarded rather as incidental, than essential to dilatation.

It may be inquired how, in this case, the pulse was weak, while the action of the left ventricle was increased. This apparent anomaly is of frequent occurrence; and, what is still more remarkable, it may take place in cases of hypertrophy as well as of dilatation. In fits of asthma or great dyspnœa for example, the pulse is often scarcely perceptible, while the heart is felt to be in a violent tumult. In other cases, both the impulse and pulse are diminished, and nothing is then felt in the præcordial region, but an obscure, profound, rolling or fluttering motion.

The inference from these facts appears to be, that when the heart is congested beyond its propulsive power, its efforts are expended on itself, without communicating strength to the pulse;* and that when the engorgement is extreme, its muscular power is more or less paralized or suspended.

In addition to its other qualities, the pulse, in the above case, was later than the ventricular systole. I have found this to occur in nearly all conditions of the heart in which the blood was propelled with difficulty, but especially in dilatation, and in contraction of the mitral valve.

The hydro-pericardium was indicated by the undulatory nature of the impulse; by its want of coincidence with the sound of the ventricular contraction; by the sensation, communicated through the stethoscope, that the heart did not strike the ribs

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* Vid. page 63 for an explanation of this phenomenon.

immediately; and by the extensive dulness of the præcordial region on percussion. These, according to my experience, are the best physical signs of hydro-pericardium; and when supported by general signs they will rarely, I believe, be found fallacious.

The polypi evince the difficulty with which the boold was circulated through the heart, these formations commonly being results of its stagnation. Their organised appearance and internal softening afford reason to believe that they had existed for a considerable time. The case presents an instance of failure of the vital powers on the disappearance of dropsy,—a common event in aged persons, or exhausted constitutions.

CASE VIII.

Simple dilatation of the heart, with softening; pleurisy.

Patrick Gillan, æt. 43, a hawker, admitted into St. George's Hospital, under Dr. Chambers, June 24, 1829. Pain in the left hypochondrium with inexpansibility of the ribs over the part affected; slight cough; scanty, white expectoration; decubitus easiest on the side affected; pains in the head and shoulders; orthopnœa; difficulty of respiration increased by ascending; pulse 80, feeble, irregular, and extremely intermittent; skin cool;

tongue furred and yellow; bowels costive; urine high-coloured. Turgescence without pulsation of the jugulars.

A fortnight before admission he was seized with general rheumatic pains, stitch in the left side, and dry cough. During the preceding winter he had vomited two quarts of black blood intermixed with food.

(V. S. ad ℥xij—℞ Haust. salin: cum sulph. potassæ ʒi, 4^{tis} horis. Diæta parciss:)

Auscultation.—No *impulse* that raises the head, (applied to the cylinder,) but a fluttering motion, with an occasional shock of some strength. A short, flapping first sound, not much louder than natural. No bellows-murmur of either sound.

Diagnosis.—Passive dilatation of the heart; no ossification of the aortic valves, nor disease of the mitral according to the evidence of auscultation, though the irregular, fluttering action, and feeble, intermittent pulse, favour the idea of regurgitation into the left auricle.

Œdema of the legs with scanty urine supervened. Diuretics and purgatives with camphor and hyoscyamus were prescribed and the emplast. belladonnæ was applied over the heart. The effect of the medicines was satisfactory, but the constitution was worn out, and he died a month after admission.

Autopsy.—The heart was dilated to nearly double its natural size; the parietes were about natural, or attenuated if either, but they were very flabby, and had externally a leucophlegmatic or infiltrated appearance and feel. *Valves* and aorta were sound. *Lungs* were gorged with blood and serum and contained a few isolated tubercles. The surface of the inferior lobe on the left side, and the corresponding extent of the pleura costalis were covered with old, whitish lymph, which by its adhesions formed a sac, enclosing a pint and a half of serum.

Remarks.—The feeble and intermittent pulse in this case, led some to suppose that there was disease of the valves. The case itself, (and it is by no means a solitary one) proves, that such a pulse may exist totally independent of valvular disease when the debility of the heart or of the constitution is very great.

The softened state of the organ contributed to render its action more feeble and irregular, and its sounds weaker, than might otherwise have been anticipated from such a degree of dilatation. Turgescence without pulsation of the jugular veins, as in this case, is very characteristic of a dilated or otherwise enfeebled right ventricle. When not enfeebled, and especially when hypertrophous as well as dilated, the turgescence is always accompanied with pulsation. As congestion of the venous capillaries predisposes to hæmorrhage, it is probable that the hæmatemesis which occurred during the previous winter, was attributable to this cause. The death of the patient was accelerated by the pleurisy, and not only by its direct effect, but by its hurrying the action of the heart and increasing the embarrassment of the circulation. It is, indeed, generally by accidents of this kind that diseases of the heart are brought to their fatal termination, and this fact suggests an important practical lesson—that, in persons affected with organic disease of the heart, all complaints capable of hurrying the circulation, and especially those of an inflammatory nature, should be regarded and treated as maladies of serious importance, capable of suddenly and unexpectedly producing a series of the most dangerous effects.

CASE IX.

A small heart with thin left ventricle; the right ventricle slightly dilated; enormous enlargement of the liver, displacing the heart; enlarged and granular state of the kidneys.

Maria Quin, æt. 28, married, of delicate fibre and complexion, was admitted into St. George's Hospital, under Dr. Chambers, August 19, 1829, with enormous œdema of the lower extremities; a tumor, very tender on pressure, in the right hypochondrium—obviously an enlargement of the liver; abdomen otherwise scarcely swelled; decubitus on either side, but cannot lie very low; dyspnœa and palpitation on ascending; jugular veins turgid, and slight undulation; pulse 96, soft, small and regular; skin natural; tongue whitish; bowels relaxed; urine Oiij or iv or more in twenty-four hours; catamenia suppressed for two years and a half; has not been pregnant for seven years.

Had been "short-winded" from infancy, but her health was otherwise good until three years ago, when she had a severe rheumatic fever, which deprived her of the use of her limbs for nine months, and was attended with great pain across the scrobiculus cordis, without cough. From that time the dyspnœa became worse. About fifteen months ago œdema of the ankles supervened; and the enlargement in the right hypochondrium was perceived only three weeks before admission.

Auscultation.—Impulse of the heart almost entirely absent. *Sounds* are very flat, clear and short, especially the second; they are louder near the left mamma, than on the sternum; and are audible on the left scapula, and very feebly on the right.

Diagnosis.—Dilatation of the heart; no adhe-

sion of the pericardium. Enlargement of the liver. Hirud. xij hypochond. dextro.—℞ Pil: Hydr: gr. iij, Scillæ gr. i, Digitalis gr. ss, Pil ter die.—℞ Inf. aurant. C. ʒx, Potassæ supertart. ʒi, Sp: ætheris nitric. ʒss, Acasiæ pulv. Mannæ āā ʒi, m. f^{t} haust. ter die.—Diæta ordinaria.

The tenderness of the liver was removed by the leeches, and six or eight watery stools and a very copious secretion of urine were occasioned by the medicines. In the course of five weeks the dropsy was nearly gone, but she had an attack of erysipelas which proved fatal.

Autopsy.—Cavities of the pleura contained a pint of fluid. *Lungs* healthy in structure and not much gorged, but they had a doughy feel and appearance. *Liver* enlarged to nearly double; surface uneven or undulating; inferior margin thick and round and internal structure slightly granulated. The *spleen* natural. *Kidneys* twice the natural size, and had a flaccid feel and infiltrated appearance. Their surface was mottled with pale spots, presenting a granular aspect. The cortical part, when displayed by a section, resembled adipose matter thickly interspersed with granules about the size of a mustard seed, of an opake and lighter yellow colour, blending softly with the more transparent fatty ground. This condition has been ably described by Dr. Bright, p. 67-9.

It constitutes his second variety or degree. Surrounded by this texture the tubular portions were seen of a dark, but dim red colour. *The heart* was displaced a little to the left by the enlarged liver. It was unusually rounded, being short in proportion to its breadth. The right ventricle was dilated to about a third more than its natural capacity. The left ventricle was not dilated, but its parietes were attenuated, being scarcely more than one-third of an inch thick. No positive opinion could be formed as to the size of the auricles, but they were considered dilated, if either. The muscular substance was firm and red.

Remarks.—The short, flat and clear sound of the ventricles, with the deficient impulse, and a soft, small pulse, denoted the dilatation. Though the left ventricle was not enlarged, its parietes were attenuated, which has an effect tantamount to dilatation in producing the sound in question. The sounds were more audible in the region of the left ventricle than in that of the right, in consequence of the heart being displaced to the left by the enormous enlargement of the liver, which extended entirely across the epigastrium. If the displacement had not been taken into consideration, it might have been supposed that the dilatation was on the left side alone. This source of fallacy, therefore, is not to be overlooked in judging which is the side of the heart affected.

This case presents several other interesting features. The heart was small in proportion to the body ; for though it was dilated laterally, its longitudinal diameter was deficient. This disproportion had probably existed from infancy and occasioned the habitual shortness of breath, and the delicacy of fibre and complexion. It is an instance of the heart with naturally thin parietes, which is found by experience to be attended with constitutional delicacy, both nervous and muscular, and to constitute a predisposition to dilatation. Though the dilatation was so slight as not to be indicated by general signs, it was rendered very certain by those of auscultation. As the sound of dilatation existed equally on the left side of the heart, although the left ventricle was merely attenuated without being dilated, it follows that the sound in question is dependent on the thinness of the walls.

Such a state of the heart as existed in the present case is not necessarily a direct cause of dropsy. A slight shortness of breath was the only inconvenience resulting from it up to the age of twenty-five. But it favors the operation of other causes in establishing congestions of the liver, lungs, or other viscera, which by further impeding the circulation, re-act upon the heart, increase its inability to propel the blood and thus occasion dropsy. There was here a co-operation of three causes—the state of the heart, the liver, and the kidneys. The good effect of hydrogogue cathartics, when the patient is sufficiently strong to bear them, was strikingly displayed in this case. By six or eight watery evacuations daily and a copious secretion of urine, the œdema of the legs, originally enormous, was in four days almost completely reduced.

CASE X.

Enormous dilatation and extreme extenuation of the left ventricle; dilatation and hypertrophy of the right; pulmonary apoplexy; enlargement of the liver.

William Lambert, æt. 52, an eating-house keeper, tall, emaciated, of exsanguine, sallow complexion, was admitted into St. George's Hospital, under Dr. Chambers, September 6, 1829, with pain in the chest, principally at the base of the sternum, and increased by full inspiration. Cough; expectoration copious, viscous, deeply coloured with blood; dyspnœa with cough in agonising paroxysms, induced by any exertion, particularly ascending; the right jugulars slightly tumid, with pulsation; fluctuation of the abdomen; slight œdema of the legs; enlargement and induration in the region of the liver; decubitus easiest on the right side. Pulse 70, intermittent, rather weak, sometimes scarcely perceptible: skin cool, tongue furred, of cream colour; bowels costive; urine high coloured and scanty.

Ill nine months. Complaint began, (after protracted mental anxiety) with cough and dyspnœa, which frequently occurred in paroxysms. The ascites had existed, more or less, for two or three months previous to admission; and the œdema of the legs for a week only.

Auscultation.—The inferior dorsal region of the chest, on the right side, is dull on percussion, and has a slight crepitant râle. The superior lobes of the lungs are resonant, but the respiratory murmur is puerile and bronchial. The *impulse* of the heart is slightly tumultuous or confused, but very feeble. *Sounds* are little louder than natural, but the first is short, like the second: they are audible at the clavicles, especially the right.

Diagnosis.—Peripneumony or pulmonary apoplexy of the right lung; bronchitis; dilatation of the heart; (particularly the right ventricle?) enlarged liver.

Cucurb. cruent. inter scapulas ad ℥xij ℞ Inf. Rosæ ℥iss, magnes. sulph. ʒij, sp. ætheris nitric. ʒss, 6tis horis.—℞ conf. sennæ ʒi, potassæ supertart, ʒss, omni nocte. Diæta lactea. The symptoms were alleviated at first; but effusion was found, by auscultation and percussion, to increase rapidly in the right pleura, and the cough and dyspnœa suffered a corresponding aggravation. When the circulation was accelerated, the action of the heart was occasionally found to be more vigorous than natural, though the pulse, at the same time, continued feeble and small, but tolerably regular. The sputa maintained their deep muddy red stain to the last. Orthopnœa with the utmost distress from a sense of suffocation became constant, and the patient expired five weeks after admission.

Autopsy.—The right cavity of the chest was filled with clear, chlorine-coloured serum; and the lung, compressed against the spine, was reduced to the size of a spleen. The pleura pulmonalis was covered with lymph, in honeycomb reticulations; and the pleura costalis was mottled with patches of red vascularity. The compressed lung felt doughy and non-crepitant. The margin of the lower lobe was in the second degree of hepatization, bordering on the third, a little pus exuding on pressure. In the midst of this, was a mass of pulmonary apoplexy, as large as an egg, claret-coloured, granular, of great density, and bounded abruptly by a wall of straw-coloured lymph. Similar masses existed in the other lung, with sanguineous engorgement, but no hepatization. *Heart.*

The *left ventricle* was dilated to a capacity which would easily contain the largest orange, or even a small melon. The parietes did not anywhere exceed a quarter of an inch in thickness, and throughout the lower half they varied from one to two lines. Over a small extent, near the apex, the muscular substance was totally deficient, and the membranes alone formed the barrier. At this part, however, the pericardium had been thickened and strengthened by an external layer of lymph,—as takes place over large vomicæ contiguous to the pleura, and which we cannot but regard as a wonderful provision of nature to obviate sudden death, which must otherwise so frequently occur. Many large coagula of bloody fibrine lined the cavity, and adhered tenaciously to the columnæ carneæ. The *right ventricle* was dilated, but to a rather less extent than the left, and its parietes were in parts four or five lines thick. Both *auricles* were dilated. *Valves* were all sound. *Aorta* was slightly dilated, but otherwise healthy.

Remarks.—The feebleness of the heart's action, the brevity of the first sound, the weakness of the pulse, and the general symptoms of venous retardation, indicated the dilated and debilitated condition of the heart. The occasional intermissions of the pulse, and the somewhat tumultuous or confused nature of the impulse might have led to a suspicion of disease of the mitral valve and regurgitation into the auricle: but in such cases the pulse is not only intermittent, but unequal and irregular, and it becomes remarkably so towards the fatal termination. These, however, were not its characters in the present instance; and as there was not, moreover, any bellows-murmur accompanying the sounds, valvular disease was excluded from the diagnosis. A sufficient cause for the intermittence and unsteady impulse, existed in the extreme degree of dilatation, and the consequent labour of the heart to propel its unnatural burden. The increased impulse of the heart when the circulation was accelerated, proceeded from the thickened state of the right ventricle; and to the same circumstance, together with the retardation in the left ventricle, is the pulmonary apoplexy to be attributed. This affection was indicated by the crepitant râle and the bloody sputa. It was the latter, however, which formed the diagnostic sign; for the stain of blood was redder, and persisted more unchanged to the last, than occurs in peripneumony, in which affection, the sputa, though pinkish at first, soon became rust or fawn-coloured, and even this stain gradually decreases as the disease advances to its resolution, or degenerates into purulent infiltration. When, therefore, such a state of the expectoration as existed in the present case, accompanies signs of obstruction on the left side of the heart, especially if conjoined with those of increased action of the right ventricle, pulmonary apoplexy may be anticipated. The tenacious coagula adherent to the left columnæ carneæ evinced that the circulation through the ventricle had been languid. These formations, when they choke the cavities, cause a remarkable aggravation of dyspnœa, and by this they may often be recognised for a week, or even longer, before death, the patient having a constant agonizing feeling of imminent suffocation. The enormous size of the left ventricle caused it to occupy a more centrical situation than natural. Its sounds, therefore, being more audible at the base of the sternum, than in the left præcordial region, the dilatation was supposed to be greater on the right, than on the left side. Laennec experienced and pointed out this source of fallacy.[*] Its rarity renders it unimportant. The pulsation of the jugulars is to be referred to the hypertrophy of the right ventricle; and the enlargement of the liver probably found its origin in congestion resulting from impeded circulation through the heart.

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[*] Tom. ii. 507.

CASE XI.

Hypertrophy and dilatation of both ventricles ; dilatation of the aorta ; roughness of its interior.

Henry Macearl, æt. about 45, an old soldier, tall, meagre, sallow, and livid when cold, was received into St. George's Hospital, under Dr. Seymour, October 28, 1829, with orthopnœa ; impulse of the heart stronger, lower, and more extensive than natural ; occasional pain in the left side, when attempting to lie upon it ; somnolency ; languor ; no dropsy ever ; pulsation of the carotids ; pulse 116—a jerk, followed by full, strong and vibrating tension,—regular ; tongue white ; bowels open.

Eighteen months previous to admission he received a kick from a horse on the præcordial region, which gave rise to his complaint.

Auscultation.—*Resonance* of the præcordial region is rather dull : that of the chest elsewhere, good. *Impulse*, very powerful above the clavicles, especially opposite to the arteria innominata. It is accompanied with purring tremor, and a loud, hoarse, abrupt, bellows-murmur ; which, when traced downwards along the sternum, becomes more hissing, and as it were, superficial. It retains the same characters, though somewhat stifled, in the præcordial region, where it drowns the natural sound of the ventricular contraction.

The *impulse of the heart* is much stronger than natural, and is followed by a vigorous back-stroke.

Diagnosis.—Hypertrophy and dilatation of the heart: dilatation of the aorta; disease of its internal coat, from the aortic valves to beyond the arch. Fiat V. S. ad ℥x, ægro recumbente. ℞ Tr. opii ♏ xxx, mist. camph. ʒx. Diæta lactea.

In the course of ten weeks he was sparingly bled six or seven times, as he stated it to be "the only thing that afforded him relief." He also took various formulæ of opium, æther, Infus. Digitalis, Ext. Lactucæ, and aromatics; but they had little effect, and he progressively declined. Œdema of the lower extremities supervened, with constant orthopnœa; pain in the præcordial region; ghastly paleness, without lividity, of the face; frequent paroxysms of dyspnœa; and extreme anxiety and distress. He died Jan. 15, 1830.

Autopsy.—The left ventricle was three fourths of an inch thick, and its cavity was dilated to one half more than its natural size. The right ventricle was equally dilated, but only slightly thickened. *Valves* were healthy, except that the aortic were a little cartilaginous, but perfectly flexible. *Aorta* was somewhat dilated; and the whole of its inner surface, from the valves to beyond the arch, was rendered extremely rough by steatomatous or cheese-like degeneration, deposited in great abundance. Patches of the same were found as low as the pelvic divarication.

Remarks.—The violence of the heart's action, and the strength of the pulse, left no doubt of the hypertrophy: and the dilatation was denoted by the extent of the impulse, the deficient præcordial resonance, and the fulness of the pulse. The dilatation and roughness of the arch of the aorta were indicated by the impulse, sound and tremor above the clavicles; and the ascending aorta was presumed to be in the same state, from the existence of the same sound along its course, only more hissing from the greater contiguity of the artery to the ear, and the interposition of a less resonant medium. It is apparent from this case, that a murmur generated in the aorta, may extend to the heart and obscure its sounds. Caution is, therefore, requisite not to mistake it for a result of valvular disease; and the distinction is easily made by observing that it is loudest above the middle of the sternum and decreases on descending; whereas, that from valvular disease is louder lower down, according to the situation of the valve affected, and decreases on ascending. When both co-exist, they may be recognised by becoming louder on approximating towards both their sources, and by some discrepancy in their nature. The vigorous back-stroke is to be remarked as a concomitant of hypertrophy with dilatation, the purring tremor of the pulse, as a consequence of powerful propulsion of the blood through a rugged aorta, and the extreme severity of the dyspnœa, as a result of the complication of disease of the aorta with that of the heart, leading to a great degree of spasmodic bronchial constriction. This was in short, one of the worst cases of cardiac *asthma.*

Case XII.

Great hypertrophy with dilatation of the left ventricle; ossification of the aortic valve; chronic pericarditis; hemiplegia and apoplexy.

Richard Porter, æt. 52, a cook, of small stature, pale, emaciated, was admitted into St. George's Hospital, under Dr. Hewett, April 8th, 1829, with hemiplegia of the left side; mouth distorted to the right, but partial paralysis of both sides of the face; a sensation of fullness and tightness about the inferior part of the sternum; cough; starting from sleep in a fit of palpitation and suffocating asthmatic dyspnœa; anasarca; pulse 96 full, and tolerably firm and regular.

Ten years before admission he had apoplexy and hemiplegia of the left side, which disabled him for half a year. He then resumed his work as a cook, and prosecuted it until three weeks ago, when he took cold, and became affected with anasarca, to which he had been subject.*

Auscultation.—July 2. Very loud rasping-murmur. (A momentary examination.) *Diagnosis. Disease of the valves of the heart.* He died July 10th, in consequence of a fit of apoplexy.

Autopsy.—Head. A small coagulum of blood under the dura mater, at the vertex of the brain, and three or four ounces of serum at the base. *Chest.* In the cavities of the pleura were upwards of three pints of serum; and in the *pericardium* was above a pint, deeply coloured with blood. The whole interior of the sac, and the surface of the heart were invested with a thick stratum of shaggy, and highly vascular, reddish lymph. *Heart.* The left ventricle was thickened to almost double —or to nearly an inch; with general dilatation of the heart. *Aorta.* Its internal membrane was slightly corrugated by steatomatous degeneration, intermixed with a few calcareous scales. *Valves.* The edge of one of the aortic valves was encumbered with an osseous concretion as large as a pea, of an elongated form, projecting into the artery, and with an irregular, denuded and scabrous surface.

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* With this account of the early history I was favoured by Dr. Hewett, under whose care the patient was admitted. I did not see him till July 2.

Remarks.—Though the detail of this case is defective, it is, notwithstanding, one of great practical value. It demonstrates that a very considerable impediment in the aortic valves does not necessarily prevent the pulse from being full, and tolerably firm and regular; the reverse of which was believed by the older writers. It shows that a scabrous ossification not only occasions a loud murmur, but one of a rasping or grating character. The case, furthermore, presents one of the numerous instances of palsy or apoplexy connected with hypertrophy of the left ventricle; and, as an interval of ten years had elapsed between the first and second paralytic attack, during which he had continued at his accustomed avocations, it shows with what an extent of disease of the heart the functions of life may be maintained. Steatomatous and calcareous disease of the aorta is so frequently accompanied with hypertrophy of the left ventricle, that it is natural and rational to regard the latter as a result of the obstacle to the circulation presented by the former. But, on the other hand, the frequent occurrence of the same disease in the arteries of the brain when the left ventricle is hypertrophous, leads to the inference that over-distention may occasion it, and, consequently, that its existence in the aorta may sometimes be secondary to the hypertrophy of the ventricle. On either view, the diseases described of the aorta and of the heart respectively, are cause and effect, and hence, the practical deduction is, that, when either exists, it is requisite to keep the circulation tranquil, in order to prevent the developement of the other.

The chronic pericarditis probably existed before his admission, and occasioned the sensation of fulness and tightness about the inferior part of the sternum. It is not unusual to find bloody fluid effused by organised lymph of the pericardium, especially when, as in the present instance, this membrane is in a state of sub-acute inflammation.

CASE XIII.

Hypertrophy; (simple;) contraction of the aortic valve to the size of a small pea; asthmatic fits about noon daily.

Wm. Hedgley, æt. 10, was admitted into St. George's Hospital, under Dr. Hewett, April 17, 1830, with respiration very hurried; temporary pain and constriction in the præcordial region; extensive pulsation of the heart; slight cough; œdema around the eyes; daily febrile accessions with palpitation, coming on about noon, and consisting of chilliness for an hour, heat for half an hour, and perspiration till evening; pulse 120 very small, weak and unequal; tongue thickly furred, moist; skin cold, perspiration; bowels regular; urine scanty, dark and thick.

Did not complain until seven weeks ago, when the paroxysms, accompanied with pain at the heart, first attacked him.

Auscultation.—Resonance of the chest natural. Impulse of the heart increased. Sound of the ventricular contraction is that of sawing. (bruit de scie.) He died on the 11th.*

Autopsy.—Walls of the *left ventricle* were upwards of half an inch thick, and very firm; those of the *right* were slightly thickened; both cavities were about natural. The *aortic aperture* was contracted by fibro-cartilage to the size of a small pea. Two ounces of serum in the pericardium, and six in each pleura. Lungs, at the lower parts, were congested and somewhat condensed.

Remarks.—The disease of the valve was clearly indicated by the sawing-murmur, and the hypertrophy by the increased impulse. The case proves that an extreme degree of contraction of the aortic valves renders the pulse small, weak and unequal. The intermittent febrile paroxysms must perhaps be attributed to congestion of the heart, exciting its reaction; and the concomitant pain can only be referred to the same cause. Why the attack occurred at the same hour daily, is not very apparent, unless it can be accounted for on the principle that the animal frame displays a general tendency to perform many of its functions in a circle, or at equal intervals. In the case of May, the paroxysm occurred at the same hour every night.

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* I was favoured with the notes of this case by Dr. Hewett, as I did not see the patient until the post-mortem examination.

CASE XIV.

Dilatation of the heart: ossification and slight dilatation of the ascending and descending aorta; dilatation of the bronchi; hydro-thorax.

Richard Storer, æt. 73, feeble and decrepid, was received into St. George's Hospital, under Dr. Seymour, July 8th, 1829. His symptoms were, palpitation; dyspnœa, aggravated by the slightest exertion; respiration extremely wheezing; cough; copious expectoration; universal dropsy; jugular veins turgid without pulsation; pulse 90, full, strong and tense.

Subject to a chronic cough for fourteen years. Swelling of the face came on ten weeks before admission, and was followed by that of the feet, scrotum, &c.

Auscultation.—Slight pulsation and soft bellows-murmur above the clavicles; *impulse* of the heart not perceptible to the hand, and it can only be felt occasionally by the cylinder. Its power is then considerable, but it is rather a blow, than a heaving of the thoracic parietes. *Sounds*—both are short and flat; neither is very loud, but the second is the louder. Excessive mucous râles in the chest, which obscure any murmurs of the heart. (Bruit de soufflet was distinguished at a subsequent examination by Mr. Johnson.) At the lower part of the left scapula there is loud pectoriloquy and gurgling râle.

Diagnosis.—Dilatation of the heart; no aneurism, nor appreciable dilatation of the arch of the aorta. Hydro-thorax and dilatation of the bronchi on the left side.

℞ pil. Hydr. gr. iij, pulv. scillæ gr. i. pil. ter die sum.—℞ potûs potassæ super-tart. Oi quotidie—℞ elaterii gr. ss, hydr. submuriat gr. ij, alterno quoque mane sumend.

In three weeks the dropsy was greatly reduced, but, as the legs continued œdematous, slight incisions were made in the calves, by which the fluid was evacuated. After this, his strength gradually failed, and he sank in four days.

*Autopsy.**—Heart was very large. All its cavities were dilated. The parietes of the left ventricle were about natural, or perhaps thicker. *Valves* healthy; but there were slight calcareous depositions beneath the bases of those of the aorta, and under the internal membrane of the heart, between the aorta and the mitral orifice. *Aorta.* No dilatation of the arch; but some in the ascending portion, immediately before the branches; and again, beyond the origin of the left subclavian. Osseous depositions, underneath the lining membrane, were scattered generally throughout the aorta and great branches; and, at the mouth of the left subclavian, a denuded patch was found. *Brain.* The arteries were diseased; especially, the basilar, which was very large and rigid. *Lungs.* The left cavity of the chest contained upwards of a pint of fluid; and the lung, compressed and collapsed, was imperfectly crepitant, and so dense as to sink in water. This condition was most marked, opposite to the inferior half of the scapula; to which part, and above, the lung was inseparably adherent. The bronchus entering this portion of lung divided into many large branches; all of which were drawn, by the adhesion of the pleura, into close apposition with the thoracic parietes; and one, not larger than a writing quill, was dilated at its extremity to the dimensions of a small nut. The *left lung* was œdematous above; and congested with blood below.

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* For the account of this dissection I am indebted to Mr. Johnson.

Remarks.—The signs of dilatation, were, the feeble impulse, and the short, flat sound of the ventricular systole. The more vigorous impulse occasionally felt, and the strength and tension of the pulse indicated that the muscular power was still considerable: in other words, that the walls of the left ventricle were not attenuated. In a young and robust subject, such a heart produces increased impulse, as in the case of Dolan, XV. The remarkable wheezing of the respiration, led to the suspicion of aneurism or of great dilatation of the arch of the aorta, these affections sometimes producing that symptom by pressure upon the trachea. The contra-indications were, the want of strong pulsation, purring tremor, and loud rasping sound above the clavicles. The slight impulse and murmur which existed there, were owing, the former, perhaps to the throbbing of the subclavians; the latter, to the ossification of the interior of the aorta and the dilatation below the innominata. The dilatation of the ascending aorta might have been recognised by tracing the murmur down the sternum, had not the loudness of the pulmonic râles rendered this impossible. Dilatation of the bronchi was inferred, because, as he exhibited no signs of phthisis, the pectoriloquy and gurgling râle could not be attributed to vomicæ. The idea was, further, countenanced by his having been subject to a chronic, asthmatic cough for fourteen years when at an extremely advanced age: circumstances peculiarly favourable to the production of bronchial dilatation. Disease of the cerebral arteries may here be remarked as accompanying enlargement of the heart. The effects of the elaterium were good; but it is a remedy which cannot be given with impunity to subjects so old and enfeebled as the present without constant watching and great discretion on the part of the practitioner.

CASE XV.

Dilatation of all the cavities, with natural thickness of the parietes; vegetations of the left auricle and mitral valve, causing regurgitation; superior cuspis of the mitral valve across the aortic orifice; contraction of the aorta.

John Dolan, æt. 28, a servant, of robust frame and pale, delicate complexion, was admitted into St. George's Hospital, under Dr. Chambers, May 27, 1829, with palpitation, increased on exertion; orthopnœa; cough; thick, white sputa; decubitus on either side; undulation of the jugulars; slight œdema of the legs; pulse 110, small, and very weak; bowels regular.

Five weeks before admission, he took cold while travelling, and was seized with pain at the heart, and cough. He was bled, and a few days ago cupped, with relief. Œdema has only existed a week. Had rheumatic fever two years ago, and several times previously.

Auscultation.—Resonance of the præcordial region, dull over a very large extent. *Impulse* much stronger than natural, and felt far beyond the usual limits and in epigastrio. *Sounds* are louder than natural; especially the second in the left præcordial region: the first is remarkable for a strong, but not grating bellows-murmur, most distinct on the left side.

Diagnosis.—Hypertrophy and dilatation of the heart; dilatation of the left auricle; obstruction, probably cartilaginous, in the aortic orifice; if osseous, the bone is not denuded. Emplast. Lyttæ regioni cordis.—℞ Haust. salin., T^{r} Hyoscy. ʒss, 6^{tis} horis.—Diæta lactea. He subsequently took, in various formulæ, calomel, haust. sennæ, sp: æth. nitric, T^{r} Digitalis, et acet. potassæ. The emplast. opii was applied over the heart. But, in a fortnight, the œdema and ascites had made progress; and in another week he was confined to bed, with constant drowsiness and profuse perspiration, which, in two days, were followed by extreme intumescence of the face. These symptoms persisted five or six days more, when he became incoherent, stupid, and, finally, comatose; in which state he expired, June 29th.

Autopsy.—Both ventricles dilated. Walls of natural thickness. Both auricles also dilated; the left, to more than double, and its interior is covered, over an extent of two square inches, with small cauliflower vegetations. These likewise pervade the whole of the mitral valve, and the chordæ tendineæ, rendering the margins of the valve so thick and knotty, as to prevent them from closing accurately. The closure is further impeded by contraction of the chordæ. The superior cuspis of the valve is displaced in such a manner as to extend across the aortic orifice and obstruct the egress of the blood. Mitral orifice, from the auricular side, expands perhaps too widely in consequence of the dilatation of both cavities. *Aorta.* Valves healthy, but the artery is remarkably contracted throughout, and, half an inch in front of the left subclavian, it is corrugated. *Lungs* œdematous and gorged with blood. Two small portions intensely dark, granular, and so dense, as to sink quickly in water (pulmonary apoplexy). *Brain* contained an ounce of serum; and *pericardium*, half an ounce. *Kidney*, large and pale.

Remarks.—This case proves, that, if dilatation be accompanied with a natural thickness of the parietes, it produces the symptoms of hypertrophy:

viz. increased action. This holds true, however, only in reference to young or robust subjects,—not to the old, or otherwise enfeebled; (as Storer). The great degree of the enlargement was indicated as well by the extent of the impulse, as of the dulness on percussion.

The murmur which attended the ventricular contraction, was occasioned, not only by the position of the cuspis of the mitral valve across the aortic orifice; but also by the patescence of the mitral valve itself, and the consequent regurgitation into the auricle. The second sound was not accompanied with murmur, because the valve expanded widely from the auricular side; and the sound itself was unusually loud apparently because the extreme distention and enlargement of the auricle, gave increased impetuosity to the influx of blood into the ventricles. The murmur was more distinct on the left side, because it there resulted partly from the mitral valve. When occasioned by the sigmoids *alone*, whether of the aorta or pulmonary artery, it is loudest about the centre of the sternum. The regurgitation together with the aortic contraction, accounted for the smallness and weakness of the pulse; and the retardation of the blood, thus occasioned, led to the dilatation of the left auricle, and eventually to that of the right cavities. The increased action of the right ventricle, conspiring with the obstruction on the left side, occasioned the engorgement and apoplexy, of the lungs. The drowsiness terminating in coma, is to be attributed to venous congestion, of which the sudden infiltration of the face was an indication. This congestion was probably increased by the extreme engorgement of the lungs; and its fatal consequences display the formidable nature of a complication which peculiarly favors such congestion: viz. increased power on the right side of the heart, and an obstruction on the left.

The murmur was judged not to proceed from denuded ossification, because the sound was less grating than is produced by bone. It is very probable that inflammation of the internal membrane of the heart and aorta, occasioned by the frequent rheumatic fevers, was the cause of the vegetations of the heart, and the puckering and contraction of the aorta.

CASE XVI.

Hypertrophy and dilatation; dilatation and pouches of ascending aorta; slight contraction of mitral valve. Hydro-pericardium. Hydro-thorax of the left side, displacing the heart to the right.

Mary Rowe, æt. 35, married, with livid lips and a circumscribed flush of the same colour on the cheeks, elsewhere sallow, was admitted into St. George's Hospital, under Dr. Chambers, Aug. 12, 1829, with orthopnœa; able to recline half-raised on the left side, but feels suffocation on the other; cough; ascites; liver enlarged; universal anasarca; undulation of the jugulars, and pulsation of the carotids; starting from sleep; pulse 110, with a full, tense swell, and considerable power; but the artery is not large; tongue white; bowels open; urine scanty and high.

Six months before admission, she fell and struck the scrobiculus cordis against a flower-pot. Intense and constant pain immediately ensued, and on the following day she was bled and confined to bed. Recovered slowly and with the supervention of palpitation and dyspnœa, for which she has been under medical treatment up to the present time.

Auscultation.—Impulse tumultuous and more perceptible in the scrobiculus cordis than in the left præcordial region.

Sounds.—The first commences with a flapping, which is almost drowned in a loud bellows-murmur. The latter is much stronger and more *superficial* from the middle, to the lower part of the sternum, near the mesial line, than in the left præcordial region. The *second sound* is a bellows-murmur, prolonged through the interval of repose, and, at the lower part of the sternum, louder than the first murmur. Bellows-sound and slight pulsation above the right clavicle; less of both above the left. Ægophony and dulness on percussion of left chest.

Diagnosis.—Hypertrophy and dilatation; disease of the valves; not improbable that those of the right side are affected; but the bellows-murmur, heard high up the sternum, is rather from disease of the aortic orifice or the artery above. Hydro-pericardium; hydro-thorax.

℞ Pil. Hydr. gr. iij, Scillæ pulv. gr. ij, digitalis pulv. gr. ss, ter die.—℞ Confec. Sennæ ʒi, Potassæ supertart. ʒss, summo mane quotidie.—℞ Haust. Pimentæ *c* Ext. Tarax. ʒss, sp. æth. nitr. ʒi, ter die. Diæta lactea. Calomel, elaterium and jalap were afterwards given, by which ptyalism was produced. In seven weeks the dropsy was greatly reduced, but the patient remained weak. At this time peripneumony supervened, and proved fatal in a week.

Autopsy.—Heart displaced to the right by fluid in the left pleura. Enlarged by hypertrophy and dilatation to one half more than natural. Walls of the left ventricle a little more than half an inch thick. *Pericardium* contained two ounces of fluid. *Valves.* Those on the right side were healthy. Free margin of the *mitral* converted into a fibro-cartilaginous ring a line in thickness and an inch less in circumference than the right auriculo-ventricular orifice. The aortic corpora sesamoidea slightly enlarged. *Aorta*, from the valves to the innominata, dilated to the circumference of five inches and a quarter, and it had, moreover, three or four pouches, the largest of which would equal the half of a walnut-shell. The whole interior was nodulated with opake, firm, cheese-like matter. *Lungs.* The left cavity of the chest was almost filled with serum, and the pleura pulmonalis was overspread with recent shaggy lymph. The lung was collapsed, non-crepitant and doughy. The lower and middle lobes of the right lung were hepatised.

Remarks.—The increased impulse, and firm, tense pulse indicated the hypertrophy; and the flapping of the first sound denoted the dilatation. The situation in which the murmur of the first sound was most audible, namely, along the course of the ascending aorta, pointed out this vessel as its source. Under such circumstances, it is difficult to determine by the sound *alone*, whether or not the aortic valves are simultaneously affected. For the criteria see p. 340.

The murmur with the second sound indicated disease of an auriculo-ventricular valve. Now, the heart was protruded to the right by the effusion in the left pleura; and the mitral valve consequently occupied the right præcordial region. This circumstance, together with the regularity of the pulse, threw some doubt over the situation of the valvular disease, and led to the suspicion that the tricuspid might be the one affected.

The contraction of the mitral valve was not great, the aperture being still nearly an inch and a quarter in diameter: yet both in this and in several similar cases with which I have met, the murmur was louder than is sometimes produced by an extreme degree of contraction. The supervention of pneumonia to extensive disease of the heart, always dangerous, is pre-eminently so when it occurs during the period of debility succeeding the disappearance of dropsy. In the present case, it was almost necessarily fatal; not only for this reason, but, also, on account of the collapsed state of the uninflamed lung, occasioned by the hydro-thorax.

CASE XVII.

Hypertrophy and dilatation; adhesion of the pericardium: contraction of the mitral and aortic valves. Hemiplegia.

Benjamin Payne, æt. 37, a basket-maker, of pale, leucophlegmatic complexion was admitted into St. George's Hospital, under Dr. Hewett, October 8, 1829, with dyspnœa and palpitation on every exertion, and occasioned in the night by lying in an uneasy position; cough; puffy swelling of the face; no œdema pedum at present, but is subject to it; sense of constriction across the epigastrium; pulse rather small and weak, slightly vibrating, regular now but it sometimes intermits every alternate beat; urine free.

For many years slightly short-winded on ascending. Fourteen months before admission had hemiplegia of the left side, which, though cured, left his present symptoms.

Auscultation.—*Resonance* deficient in the præcordial region, which is unnaturally prominent. *Impulse* is of a curbed or struggling nature, and is felt in epigastrio. It is an occasional shock with little heaving, and its force in general scarcely exceeds the natural standard; but occasionally it has a vigour considerably greater, and accompanied with a back-stroke. *Sounds.*—A prolonged bellows-murmur accompanies both, and the two are continued into each other. The flapping of the second is more audible on the second or third ribs than lower down. The impulse and first sound are synchronous. Above the clavicles there is a hoarse, but subdued and remote sound, and a very slight pulsation.

Diagnosis.—*Moderate hypertrophy and dilatation of the heart. Disease of the valves.* His symptoms were much mitigated by the usual remedies, particularly by occasional small bleedings: but they continually recurred in an aggravated form, and he sunk December 19.

Autopsy.—Adhesion of the pericardium. Left ventricle nearly an inch thick, and its cavity dilated to one half larger than natural. The right ventricle slightly hypertrophous, and its cavity enlarged, but not to the same extent as on the opposite side. The *mitral valve* converted by cartilaginous thickening, into a rugged, knotty ring, not more than half the natural size. *Aortic valves*, likewise thickened by knotty cartilage. Corpora sesamoidea, enlarged to the size of small peas, considerably obstruct the aperture. Interior of the aorta is slightly steatomatous, but smooth. *Lungs* do not collapse, and are of immense size from sero-sanguineous engorgement. Some fluid in the cavities of the pleura.

Remarks.—The enlargement of the heart was indicated by the prominence and dull resonance of the præcordial region, and by the pulsation reaching to the epigastrium. The hypertrophy was denoted by the occasional vigour of the shock, and by the back-stroke. The irregularity of the heart's action was attributable to the valvular disease. Although the *struggling* nature of the impulse was very characteristic of adhesion of the pericardium, the idea was discountenanced by the history, which, according to the patient's account of it, did not supply evidence of antecedent pericarditis. Disease of the valves was indicated by the murmur of both sounds. That of the first was occasioned not only by the state of the aortic valves, but probably also by regurgitation through the mitral. The murmur accompanying the second sound, resulted from the contraction of the mitral to half its size, and from its rugged state. The flapping of the second sound proceeded from the right ventricle, and it was more audible high up the chest than in the præcordial region, because, in the latter situation, it was obscured by the valvular murmurs. This was a case of cardiac asthma.

Case XVIII.

Dilatation and ramollissement of the heart; great contraction of the tricuspid, and still more of the mitral valve; slight, of the aortic. Hydro-pericardium.

Christian Anderson, æt. 42, in the Edinburgh Royal Infirmary, June 16th, 1825. Cheeks, nose, and lips purple; turgescence and undulation of the jugulars; dyspnœa, occasionally in paroxysms induced by cough or any exertion; starting from sleep, and frightful dreams; œdema of the face and legs; pulse imperceptible; urine scanty, and high.

Eighteen months before admission, she "strained herself opposite to the navel," by carrying heavy weights: hæmoptysis ensued and lasted three weeks attended with palpitation, dyspnœa, and cough.

Auscultation.—Impulse an irregular succussion or undulation of the chest. *Sounds.*—The first (at the lower extremity of the sternum) was a very loud filing-murmur, or that of obscured and subdued sawing. It commenced abruptly, with a flap, The second sound, short and flat, was so weak as scarcely to be audible. It concluded the first murmur. The same sounds existed on both sides of the heart, but were more subdued and indistinct on the left. They were more or less audible over the whole anterior surface of the chest.

Diagnosis.—Much disease of the valves; dilatation of the heart, particularly on the right side; parietes flaccid; not thickened.

Autopsy.—The heart was nearly twice its natural size. *Right auricle and ventricle* much dilated; the latter larger than an orange. Parietes of both of natural thickness, but the ventricular columnæ carneæ enlarged. Muscular substance firm but pale. *Left ventricle.* Its cavity enlarged to the size of a goose's egg. Walls of natural thickness, but pale, flaccid, and easily lacerable. *Left auricle* slightly thickened and dilated. *Tricuspid valve* an uneven thick cartilaginous ring, which admitted the middle finger. *Mitral valve* was a similar ring, as thick as a crow-quill, admitting the end of the little finger. *Pulmonic and aortic* valves were natural, except that the corpora sesamoidea of the latter were enlarged and cartilaginous, but not so as to prevent the valve from discharging its function. The pulmonary artery was somewhat dilated. *Pericardium* contained ℥vij of serum; and the cavities of the pleura about Ov or vi. *Lungs* œdematous, and slightly tuberculous.

To the original notes of this case is annexed the following remark:—" As the pulmonic and aortic valves were equal to the discharge of their function, the (filing) sound proceeded from regurgitation through the auricular valves. Hence if 'bruissement' be heard during the ventricular contraction, we are not necessarily to infer, that there is disease of the aortic or pulmonic, rather than of the auricular valves." It might be objected to this argument, that the enlarged corpora sesamoidea of the aortic valves were capable of occasioning the murmur of the first sound. To this we may reply in the negative; as the current of blood through the aortic valves was too feeble to excite a murmur, since it was incapable of creating a perceptible pulse. The greater weakness of the murmur on the left side, appears to me attributable to two circumstances:—1st, The smallness of the mitral aperture; in consequence of which the quantity of fluid retropelled, was inconsiderable. 2nd, The ramollissement of the left ventricle: whence the retropulsion of the fluid was languid. The deficient supply of blood, and the inadequate power of the ventricle, account for the imperceptible pulse. On the right side of the heart, the ventricle was stronger, and the tricuspid valve was double the size. Hence, the murmur was louder. The second sound was scarcely audible. This is what we should expect. For the scanty supply of blood through valves so contracted, would not dilate the ventricles with sufficient velocity to occasion much sound. Nor was this second sound accompanied with murmur; because, as the ventricles, in consequence of their dilatation and ramollissement, possessed little resilient power, the blood, deprived of their suction, passed indolently through the valves. The undulating motion of the heart was occasioned by the hydro-pericardium. It is to be remarked, that obstacles in front had led to dilatation of each of the cavities successively in retrograde order.

This case is interesting as proving the occurrence of murmur with the first sound from regurgitation,—a fact overlooked by Laennec. The next case proves the same.

CASE XIX.

Elizabeth Dennis, æt. about 50. Emaciated, admitted into the St. George's Infirmary under Dr. Clarke, December 8, 1830.* Affected with all the symptoms of organic disease of the heart in their most severe form. Has been affected with ascites and anasarca. Bellows-murmur accompanying the first sound in the region of the left auricle but not in that of the aortic valves. Impulse strong, pulse irregular, unequal and extremely feeble, later than the ventricular systole.

Diagnosis.—Hypertrophy and dilatation. If there is no disease of the aortic valves the bellows-murmur is from regurgitation through the mitral. Is it a ring?

Autopsy.—(Performed in the presence of Dr. Clarke, Mr. Howship, Mr. Syme, House-surgeon to the Infirmary, and the writer.) Hypertrophy and dilatation of the heart. All the valves healthy except the mitral, the free margin of which was thickened by fibro-cartilage and the chordæ tendineæ were shortened in such a manner as not to allow the pillars of the valve to come in apposition: hence a space, judged to be about as large as a finger, was left, through which regurgitation could take place.

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* My friend Dr. Clarke kindly invited me to see this case. I wrote the physical signs with the diagnosis in his journal from which I now transcribe them.

Remarks.—This case affords evidence, which will, I conceive, be considered unequivocal that regurgitation through an auriculo-ventricular valve, occasions murmur with the first sound.

CASE XX.

Aortic valves rigid; mitral, extremely cartilaginous and ossified; tricuspid, cartilaginous; great dilatation.

George Sharpe, æt. 33, sallow, with livid palpebræ, was admitted into St. Bartholomew's Hospital, under Dr. Latham, June 7, 1826. Symptoms were, great palpitation and dyspnœa, sometimes occurring spontaneously; great œdema pedum; congestion and undulation of the jugulars; somnolency; pulse 130, weak, irregular, and intermittent. Urine scanty and high.

Short-winded, so that he could not run up stairs, for eight or ten years. For three or four years has had a constant short cough, with great proclivity to bronchitis. Has been much worse since a severe cold contracted six months ago.

Auscultation.—Resonance of the præcordial region extensively dull. *Impulse*, though feeble, is felt from the fourth to the eighth rib. Below the left nipple, the shock is somewhat stronger than natural. *Sounds.* The first is a grating combined with a whizzing murmur, which, over the left ventricle, is loud and near to the ear; while, over the right, it is as if remote. In the latter situation, the flapping of both sounds is remarkably loud. The second sound, on the left side, is without murmur.

Diagnosis.—Dilatation and hypertrophy of the left ventricle, but walls not appreciably thickened. Right ventricle and auricle dilated, but not hypertrophous. Valvular disease on the left side. On the right side also? (If the event disprove this, does the murmur heard on the right side proceed from the left?)

Autopsy.—℥ij or iij of serum in the pericardium; Oij in the chest, and as much in the abdomen. *Heart* enlarged to nearly double. Right ventricle would contain a large lemon; its walls were less than one fourth of an inch thick, but the columnæ carneæ were enlarged. Auricular orifice considerably widened. Loose margin of the *triscuspid valve* cartilaginous and thickened, but it was judged capable of closing the aperture. Left ventricle would contain a small lemon; walls half an inch thick at the base, and a quarter at the apex. *Aortic valves* very rigid with cartilage. *Mitral* *valve* extremely diseased. The base and margin were of fibro-cartilage, intermixed with denuded bone. A lamellated polypus of organized lymph and as large as a walnut, grew in the auricle by vascular connection with the lining membrane, which was rough, opake, and yellow.

The internal coat of the arteries was stained of an intense red.

Remarks.—The extensive dulness, the languid impulse, and the loud flapping sound of the ventricular contraction, denoted the dilatation; while some degree of power in the shock below the left nipple, indicated that the walls of the ventricle were not attenuated. The valvular disease on the left side was denoted by the murmur. The compound nature of the murmur, partly whizzing and partly grating, indicated that both valves were affected. I have frequently met with this compound species of murmur, the whizzing character appertaining to the aortic valves, in consequence of their being nearer the surface. See p. 340. This is well exemplified in another individual at present under my notice, affected with disease of both valves, in whom there are from two to five beats of the heart accompanied with grating murmur, but no pulse in the radials: then succeeds a stronger shock with a pulse, and a hissing opposite to the aortic valves. In the present case the grating sound, the feebleness and instability of the pulse and the general symptoms of obstruction on the left side of the heart, left little doubt that there was regurgitation through the mitral valve.

Why was not the second sound, or that of the left ventricular diastole, accompanied with murmur from the contracted mitral? Probably because the old polypus in the auricle, by further obstructing the orifice, did not allow of a sufficiently copious and rapid passage of blood to occasion a murmur. See p. 57. The second sound, then, proceeded from the right ventricle.

CASE XXI.

Contraction of the mitral to the size of a pea. Dilatation with hypertrophy.

Mrs. M'Lean, Royal Infirmary Edinburgh, August, 1824. Laboured under all the symptoms of organic disease of the heart in an aggravated form. In particular, had suffocative dyspnœa, great lividity of the face, and an irregular pulse.

Auscultation.—Impulse extremely strong, and perceptible over the whole anterior surface of the chest and in the epigastrium. *Sounds* slightly increased. No bellows-murmur or purring tremor was noticed.

Autopsy.—Left ventricle moderately hypertrophous and dilated. Right ventricle would contain an orange. Columnæ carneæ and the parietes in parts thickened. All the valves natural except the mitral which was contracted into a fibro-cartilaginous ring as thick as a small goose-quill and capable of admitting a moderate sized pea. Its internal surface smooth; external nodulated. Into the latter the membranous part of the valve was inserted. The chordæ tendineæ had disappeared and the columnæ carneæ were inserted *immediately* into the ring. The auricle behind this valve was considerably dilated and greatly thickened.

Remarks.—A murmur of the second sound might have been expected to result from this valvular contraction: as it did not exist, the case perhaps tends to prove that when the contraction is very great, murmur is not occasioned or only in a slight degree.

Case XXII.

Great dilatation of the pulmonary artery. Hypertrophy and dilatation of the heart.

Sarah Wetherly, æt. 36, of yellowish complexion, was admitted into St. George's Hospital, under Dr. Seymour, January 20th, 1830, with dyspnœa; pain at the scrobiculus cordis; ascites, œdema pedum; pulse 70 large, full, and rather tense; tongue clean; urine scanty; catamenia suppressed for five months.

Short-winded for ten years, in consequence of striking her breast against a post. Eight months ago the catamenia were checked by cold, from which time she dates her complaint; but the œdema did not supervene until three months afterwards, when the menstrual flux became totally suppressed.

Auscultation.—Resonance of the præcordial region is extensively dull; prominence, pulsation, and purring tremor between the cartilages of the second and third left ribs.

Impulse, much more extensive and considerably stronger than natural, particularly in the left præcordial region. The pulsation is felt in epigastrio. *Sounds.* The first, is an extremely loud, harsh, and *superficial* sawing-murmur. It is extensively audible, but most so on the prominence between the second and third ribs.

Diagnosis.—Hypertrophy and still more dilatation of the heart—greatest on the left side. Dilatation of the origin of the aorta, probably forming an aneurismal pouch towards the left. V. S. ad ℥x.—℞ calomel. gr. iij hâc nocte.—℞ haust. sennæ cum tart. potassæ ʒiij cras mane.—℞ haust. salin. efferv. ter die.

Died a month after admission.

Autopsy.—Heart encroached much, by its size, on the left side of the chest. It was hypertrophous and dilated; most on the left side.

Pulmonary artery remarkably dilated. Its internal circumference near the valves was four inches and a half: and midway between this and the bifurcation, it was five inches. The enlargement did not extend beyond the bifurcation. The sigmoid valves appeared to be put on the stretch, and too small to close the orifice. *Aorta* rather contracted. *Mitral valve* slightly thickened. *Abdomen* contained three or four quarts of straw-coloured fluid. *Liver* rather enlarged and hardened, and its peritoneum thickened by old inflammation.

Part of the diagnosis, in this case, was inaccurate: but as dilatation of the pulmonary artery is one of the rarest affections incident to the human frame, and its signs had not ever, to my knowledge, been described; while aneurism of the ascending aorta is an ordinary disease with well known signs, the former could not, on any certain grounds, have been diagnosticated in preference to the latter.

On reviewing the signs of the former, however, they appear to me so pathognomonic as to render the affection easy of diagnosis for the future. See p. 436.

As the pulmonary artery is close to the surface, the sound possesses in a peculiar degree the character of proximity to the ear of the auscultator.

The strong impulse, and tense pulse denoted the hypertrophy. The great extent of the pulsation, the præcordial dulness, and the largeness of the pulse, indicated the dilatation. The left side was supposed to be more enlarged, because the impulse was strongest over it.

Case XXIII.

Aneurism in the substance of the left auriculo-ventricular septum. Dilatation of the heart; parietes natural.

James Brown, æt. 27, a tailor, complexion cadaverously pale, admitted into St. George's Hospital under Dr. Chambers, Dec. 9, 1829. Palpitation; vehement impulse; throbbing of carotids; œdema pedum; dysentery; pulse 130 full, strong and jerking.

Is a drinker. Has been short-winded for a year at least, and disabled for three months. Died January 15th, 1830.

Autopsy.—Left ventricle dilated, but parietes of natural thickness. *Aortic valves.* Their bases in parts detached by steatomatous disease. Beneath the valve nearest to the left auricle, the little finger could be introduced, and insinuated under the lining membrane of the heart to the extent of half an inch, when it emerged through a rugged, steatomatous opening into the cavity of the ventricle. From this canal, a second extended transversely to the left, into the muscular substance of the septum between the auricle and ventricle; and here it formed a pouch about as large as a nut, which bulged upwards and backwards, behind the pulmonary artery.

Remarks.—Real aneurism of the heart of this kind is rare. The hypertrophy was that by increased extent, without thickening of the walls.

CASE XXIV.

Sacculated aneurism and great dilatation of the ascending aorta; adhesion of the pericardium; simple hypertrophy of both ventricles.

Benjamin Thomas Hill, æt. 40, a paper-hanger, of florid complexion and sanguine temperament, was received into St. George's Hospital, under Dr. Wilson, Aug. 12, 1829. An immense pulsating tumor, extending from the third to the sixth rib, on the right side of the sternum. Its most prominent point is about the fourth rib, where there is tenderness. Pain in the right side of the chest, occasionally extending around the back and right shoulder, with a sensation of weight in the arm.

Palpitation; little dyspnœa, except on exertion; cannot be on the right, and scarcely on the left side. General health good; pulse 108, full, rather tense, and a little behind the impulse of the heart and aneurism; tongue white.

After an "over-reach," nine months before admission, the aneurism commenced merely as a pulsation. The pains and other symptoms were not felt until six months afterwards.

Auscultation.—Impulse of the tumor is exactly synchronous with that of the heart, and is violent. It decreases towards the clavicle, immediately below which it is scarcely perceptible. Above the right clavicle there is a considerable pulsation; while above the left there is very little. *Impulse of the heart* is stronger, rather lower, and more towards the left than natural. *Sounds on the tumor*. The first is a loud, hoarse, prolonged murmur, terminating in a second of the same nature, but shorter. Above the right clavicle, the first murmur is hoarser and more abrupt than on the tumor. Above the left clavicle, it has the same character, but is weaker. *Sounds of the heart*. The first is a bellows-murmur distinct up the whole length of the sternum. The second is a prolonged murmur, much weaker than the second murmur on the tumor.

Diagnosis.—Dilatation of the ascending aorta; a sacculated aneurism, springing from immediately *above the valves on the right side. Hypertrophy of the heart.*

In the course of four months, the patient was greatly benefited by low diet and great tranquillity; by the occasional application of leeches to the tumor when painful; and by wearing, in the intervals, a plaster to sooth and support it. He was then attacked with cynanche tonsillaris, and three days afterward with erysipelas of the face, which created great febrile excitement and irritation with anxiety and dyspnœa. Antiphlogistic measures were employed with the greatest care and judgment, but without effect, and he sunk at the expiration of a week.

Autopsy.—Pericardium universally adherent by thin but not close false membranes. Left ventricle nearly an inch thick: right, half an inch. *Valves* healthy. *Aorta,* from the valve to the arteria innominata, is dilated to the size of an average lemon, and its lining membrane is thickened and puckered. From the right side of this dilatation, and close above the valves, springs an aneurism, equalling a goose's egg in capacity, by a circular, abrupt-edged mouth, about two inches and a half in diameter. The walls of the sac are thin, and the lining membrane of the aorta appears to be continued throughout it, except opposite to the fourth rib, where the bone is denuded. An old polypus adheres to the base of the sac by a pedicle three-fourths of an inch thick.

Remarks.—It was certain that the aneurism sprung from the origin of the aorta, because one springing from the descending aorta, (the only corresponding situation,) would have had the lungs interposed between it and the surface. The first loud hoarse murmur heard on the tumor was occasioned by the violent irruption of blood through the abrupt-edged aperture: and the second murmur must have proceeded from the expulsion of the same blood by the elasticity of the sac and the pressure of the surrounding parts. The dilatation of the ascending aorta was indicated by the murmur audible up the whole length of the sternum, and by its being louder, and accompanied with a stronger pulsation, above the right clavicle, (to which the dilatated part was more contiguous,) than above the left. The hypertrophy of the heart was denoted by the strength of its impulse; and this was more to the left than natural, because the heart was displaced by the aneurism.

There can be no doubt that the murmurs accompanying the sounds of the heart, were, in a great measure, those of the aneurism and dilatation of the aorta propagated to the præcordial region. But, as no flapping attended the second sound, they appear to have been partly owing to the adhesion of the pericardium, which has probably the effect of cramping the action of the heart and preventing the free expansion of its orifices. The pulse was later than the ventricular contraction, because dilatation of the aorta impedes the transmission of the blood; whence also the hypertrophy of the heart probably originated.

This case presents another instance of the formidable nature of an acute complaint, supervening on extensive organic disease of the heart. The circulation, hurried beyond a certain point, seems to lose the balance essential to the maintenance of life.

The two following cases tend to show, that contraction of the mitral valve, when extreme, is not attended with murmur of the second sound.

CASE XXV.

Dilatation of all the cavities; hypertrophy of the right ventricle; extenuation of the left; great contraction of the mitral valve; ramollissement.

Mrs. —l—n, consulted me, Dec. 27, 1829. She had livid lips; a defined purplish red on the cheeks; complexion elsewhere sallow; dyspnœa and palpitation, excited even by walking across a room, and to excess, by ascending a flight of stairs; frequent cough, preventing sleep; constant copious expectoration of frothy, viscous mucus, the temporary suppression of which, by sleep or opiates, caused paroxysms of excessive dyspnœa and orthopnœa; chilliness, particularly of the extremities; universal and extreme anasarca; catamenia, regular; bowels open; pulse small, weak, unequal and intermittent; urine scanty and high; thirst; anorexia.

Complaint commenced ten years before I saw her, and was attributed to difficult parturition. The symptoms were always greatly aggravated by colds, to which she was particularly liable. She had frequently had slight œdema pedum, which subsided spontaneously. Always felt best in a warm, humid atmosphere.

Auscultation.—Impulse imperceptible. *Sounds.* Both were short, flat, and audible as far as the right clavicle. They were weaker on the left side of the heart. Murmur was not noticed. By the usual diuretics and aperients, the dropsy was completely removed in six weeks, the strength being little impaired and the appetite good. She was then suddenly seized with oppressed palpitation, suffocative orthopnœa, constant nausea, and overpowering exhaustion, anxiety and jactitation. The dropsy began to re-accumulate, the sense of suffocation became agonising, the pulse failed entirely for twenty-four hours before death, and she sunk a week after the relapse.

Autopsy.—Pulmonary apoplexy and engorgement. *Heart* double the natural size, and very flaccid and pale. *Ventricles.* Right dilated to double; its parietes were not attenuated, and the columnæ carneæ were hypertrophous. The left was less dilated, and its walls were reduced to one-third of an inch in thickness. *Auricles.* Right, dilated; its parietes thin and diaphanous. Left, greatly dilated, considerably thickened, and almost completely filled with a polypus adhering firmly to its lining membrane. *Valves.* Aortic, slightly cartilaginous, but unimpeded. *Mitral,* contracted by cartilage into a *slit* which only admitted a writing quill. ℥v of serum in the pericardium. *Liver* slightly enlarged, granular, and of yellowish brown colour.

Remarks.—This case is remarkable as presenting a degree of valvular contraction seldom if ever exceeded, and as showing with how great an amount of disease life may be prolonged for a series of years.

The dilatation was manifest from the deficient impulse, the short, flat sounds, and the general signs of venous remora. Though no murmur was noticed on the left side of the heart, contraction of the mitral valve was readily to be inferred from the small, weak, unequal, and intermittent pulse, and from the languid action of the left ventricle.

The reason why great contraction of the mitral, such as existed in the present case, should not produce murmur, is explained p. 57.

The columnæ carneæ of the right ventricle were hypertrophous. This, concurring with the obstruction of the mitral, accounted for the pulmonary congestion and apoplexy. Hence, too, the copious expectoration; which being the mode that nature adopts to unload the vessels of the lungs, it is obvious why the symptoms were aggravated when the expectoration was suppressed, whether by opiates, catarrh, or a dry, sharp air. The relapse occurred at that critical moment when the dropsy had disappeared: the sudden supervention of suffocative dyspnœa, renders it probable that the polypus in the left auricle commenced at that time, and was the cause of the symptoms and of the fatal event. Hence the importance in such cases of preventing nausea, syncope, or any affection which can cause stagnation of the blood.

CASE XXVI.

Adhesion of the pericardium: hypertrophy and dilatation: disease of the aortic and mitral valves: contraction of the aorta.

Joseph May, æt. 20, at St. George's, under Dr. Hewett, September 2, 1831, green-grocer, goes about much with heavy loads. Complexion leucophlegmatic from much puffy infiltration. Violent action of the heart, visible over the whole anterior chest, with a sense of universal throbbing, especially in the temples and vertex: action irregular: sometimes three or four unusually violent beats, occasioning vertigo and stupefaction, which caused him to sink down in a state of unconsciousness for a few seconds. Dyspnœa, greatly exasperated by any effort; until within a month, it occurred, with palpitation, in a violent paroxysm every night, compelling him to rise and lasting twenty minutes. It was always accompanied by pain in the region of the liver. An ounce of gin, which extricated flatus by eructation, never failed to relieve both the pain, the palpitation, and the dyspnœa. The attack invariably ended in a drenching perspiration and a lax dejection, followed by sleep. Had been subject to it nightly for upwards of four years, the time of its supervention being, at first, eight o'clock p. m. and becoming gradually later till it arrived at two o'clock a. m. Frightful dreams; universal dropsy; urine scanty and high coloured; pulse rather large, extremely jerking and sharp, incompressible, irregular and intermittent.

Five years ago had two or three attacks of acute rheumatism at intervals of two or three months, which left pain and palpitation of the heart. Six weeks ago nine quarts of serum were drawn off by punctures, with great relief.

Auscultation.—Impulse is *double*, forwards and backwards, with the first and the second sounds respectively, which occasions a tumultuous *jogging* motion—strongest at the left mamma.

Sounds.—Both have a prolonged filing-murmur, almost continuous, and loudest over the left ventricle, the first being the more hissing. Over the right ventricle the murmurs seem remote, while the flapping of the second sound is loud.

Above the clavicles, especially the right, strong impulse, tremor and a loud, hoarse murmur. The latter is heard of a more hissing, superficial nature along the sternum in the tract of the aorta.

Diagnosis.—Hypertrophy with dilatation of the heart, the former predominating in the left ventricle, and the latter in the right: disease of the valves on the left side; and of the interior of the aorta, with dilatation. Adhesion of the pericardium.

After being greatly benefited by the judicious treatment of Dr. Hewett, he was seized with erythema of the leg, from the excitement of which he sunk, with stupor, in four days.

Autopsy.—Heart had forced the left lung upwards to between the fourth and fifth rib, and five or six bands, half an inch long, united the pericardium to the costal pleura.

Pericardium adhered universally and closely to the heart. *Left ventricle:* walls, an inch thick; cavity size of an ordinary orange. *Right ventricle:* not thickened; dilated to double; columnæ carneæ enlarged. *Auricles* natural. *Mitral valve* thickened and opake, but not contracted or patescent.

Aortic valves.—On one was a calcareous concretion as large as a small pea, projecting conically into the centre of the artery: on another was a similar but very small deposition. The two aortic valves on the left were thickened and opake, but free.

Aorta was contracted and puckered by steatoma opposite to the left carotid, where its circumference was only two inches and a half, decreasing beyond that point.

Remarks.—The jogging action and the history indicated adhesion of the pericardium, and this caused the heart to beat higher than is usual when it is greatly enlarged. The increased impulse and extent of its range, indicated hypertrophy with dilatation. The osseous concretions on the aortic valves occasioned the loud hissing of the first sound, and that of the second proceeded from the thickening of the mitral and the relative smallness of the orifice to the immense capacity of the ventricle. The murmur above the clavicles was occasioned by the straitening of the aorta, and by the velocity with which the blood was propelled, as denoted by the extremely jerking, incompressible pulse. The impulse above the right clavicle was occasioned by the latter cause, the effect of which was probably aided by the contraction of the aorta immediately beyond. This unusual combination of circumstances led me to suspect dilatation of the aorta, which did not exist.

The regularity of the nightly paroxysms of asthma, the good effects of gin, the termination of each attack by perspiration and purging, the enormous discharge of serum by punctures, and the fatal consequences of a slight inflammatory affection, are worthy of remark.

Case XXVII.

Universal adhesion of the pericardium: hypertrophy and dilatation.

A Boy, whom Dr. Seymour obligingly took me to see in private, had universal, close adhesion of the pericardium with enormous hypertrophy and dilatation. The left margin of the sternum, over the heart, was remarkably prominent, the organ was not seated lower down than natural, its pulsation was violent and jogging, the sounds were both accompanied with bellows-murmur and the pulse was jerking. The complaint had existed for upwards of a year.

Case XXVIII.

Acute pericarditis : adhesion of the pericardium.

William Harrison, æt. 22, St. George's, under Dr. Wilson, August 11, 1830. Had extremely acute rheumatism with pain in the cardiac region, violent palpitation, and a strong jerking pulse of 110. Was repeatedly bled and took calomel and opium with temporary relief; but the pain in the heart became very intense, lancinating to the back and being increased by inspiration: the pulse became faltering and the anxiety and distress excessive. In this state he was relieved by a blister and the supervention of ptyalism. A fortnight after admission, the pulse was extremely jerking, but regular; the *impulse* of the heart was a violent smart, bounding blow, strongest at the left mamma: the *first sound* was a prolonged but not very loud bellows-murmur. The second was like a sigh made with the lips nearly closed. A month after this time, the impulse was *struggling* and strong, but not lower down than natural though the heart was enlarged. The bellows-sounds as before, but the first louder. Pulse 100 extremely jerking.

Diagnosis.—Pericarditis with effusion, terminating in adhesion of the pericardium.

This was a well characterized case. The nature of the impulse, pulse and sounds were very pathognomonic. What was the cause of the second sighing murmur? Was it inflammatory constriction of the auriculo-ventricular valves?

Case XXIX.

Pericarditis. Adhesion of pericardium: hypertrophy and dilatation.

A Gentleman, after protracted acute rheumatism, had pericarditis with pain in the cardiac region shooting to the back, and inability of lying on the sides. He was restored from an almost hopeless state by mercury and a succession of blisters. Throughout the acute stage the pulse and impulse were extremely jerking, and the sounds, especially the first, were accompanied by bellows-murmur. These characters remained for six months after the pericardium had become adherent. The heart rapidly enlarged and caused prominence of the cardiac region. He sunk at the expiration of about a year and a half.

CASE XXX.[*]

Hypertrophy and dilatation: universal adhesion of the pericardium, with double impulse. Tubercles of lungs, pleura, pericardium, heart, bronchial glands and peritoneum.

A Genevese, æt. 8, had for several months experienced frequent paroxysms of cough and become very emaciated and feeble. When visited, the symptoms were, extreme emaciation; frequent cough; easy expectoration of stringy mucus with yellowish puriform flakes; excessive palpitation; pulse very frequent.

Physical signs.—Impulse. The ear is raised by the beats of the heart which are tumultuous and of great energy.

Lungs.—Resonance of the left side in front almost flat; rather obscure behind: very clear on the right side. Mucous râle in some parts, especially on the right, where respiration is puerile. On the left, absence of respiration in the greater part of the lungs.

In two months he died from oppression and obstinate vomiting. Two days before death he had suffocative dyspnœa, with the singular phenomenon that the beats of the heart were very intense, tumultuous, and *twice as frequent as the pulse*, the heart pulsating about 150 or 160 per minute, while the pulse beat only eighty.

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[*] The two following highly interesting cases were sent to me by Dr. Lombard, a genevese physician of distinguished talents educated in England and in great estimation amongst the English residents at Geneva.

Autopsy.—The *right lung* contains crude tubercles over a great extent. Pleura sound. *Left lung* almost universally adherent to the ribs by thick tuberculous false membranes covered with albuminous flakes: less numerous but more advanced tubercles than in the right lung: some beginning to suppurate.

Pericardium universally adherent to the heart by false membranes from one to three lines thick, which contain numerous tubercles, in all respects similar to those of the pleuritic false membrane. Heart three or four times as large as natural—hypertrophy of both ventricles, but particularly of the left, of which the walls are very thick and the cavity considerable. In the substance of the walls of the right ventricle is a tuberculous tumor of six or eight lines in thickness and two or three inches in length, formed by a yellowish resistent tissue more homogeneous at the border than in the centre. Besides this principal tumor there exist several smaller, but in the right ventricle exclusively. Orifices and great vessels healthy. The base of the heart and the origin of the great vessels is encircled with a considerable mass of tuberculous glands, which completely envelope the pulmonary artery and aorta for several inches from their origin. The glands are formed of a yellow, firm, resistent, tuberculous substance: one alone is softened. By their agglomeration they form an irregular mass of several inches in diameter.

Peritoneum granular. Mesenteric glands, also liver, spleen, and intestines, healthy.

Remarks.—This case is remarkable for the prevalence of the tubercular diathesis. I have little doubt that the two beats of the heart for one of the pulse were nothing more than the impulse and back-stroke, which, as in the case of May, become very sensible and have a jogging character when there is universal adhesion of the pericardium.

CASE XXXI.

Immense aneurism of the aorta in the substance of the left lung producing hæmoptysis. Amaurosis.

Lafin, cook, æt. 58, athletic, has enjoyed good health until lately, has been seized within six weeks with head-ache and complete amaurosis of the right eye and incomplete of the left: pupils are contracted and immoveable. Complains of pain equally in the back and the shoulders, by [illegible] in [illegible]. There [illegible] and [illegible] considerable quantities of blood; [illegible] considerable quantity of [illegible] and almost [illegible]. Respiration is feeble over the whole [illegible] particularly [illegible]. Heart presents signs of dilatation of the right side. Pulse full [illegible].

He was [illegible] several times. [illegible] The [illegible] was [illegible] with [illegible] mucous [illegible] over the whole left side. Respiration [illegible] on the [illegible] of the [illegible].

Autopsy.—Extremely [illegible] to [illegible]. The [illegible] is universally adherent to [illegible] a thick [illegible] membrane infiltrated with serum. Its superior [illegible] contains an aneurismal sac of the size of a [illegible]. The sac is formed by the ascending aorta two inches above its [illegible] from the pericardium. *Its internal surface is smooth.* The internal membrane of the artery can be traced over a breadth of about two fingers. The rest of the sac is formed by the middle coat, which presents linear furrows that do not alter the polish of the surface. Outside of the sac are found fibrinous layers, less organized in proportion as they are more external: the last, which are in contact with the lung, seem formed by coagulated blood alone.

After having removed the aneurismal sac, the superior lobe of the lung is found reduced to a mere membrane composed of condensed pulmonary tissue: the air vesicles, the bronchial tubes and the blood vessels open directly on the internal surface of the covering of the aneurismal sac and are thus in immediate contact with the tumor, which, by its size has caused absorption of a great part of this lobe.

Beyond the aneurism the aorta is dilated as far as within three fingers breadth of the cœliac trunk: its internal surface is rugous over the whole of this extent and some cartilaginous points are observed in the thoracic portion.

Heart tolerably voluminous, all its cavities dilated. No contraction of orifices.

The inferior lobe of the left lung is infiltrated with pus and presents several lumps of ramollissement: some softened tubercles in that part of the superior lobe which is in contact with the tumor; none elsewhere.

The right lung is gorged with serum and presents several lumps of gray ramollissement. The bronchi are red and tumified.

The second dorsal vertebra presents a commencement of caries, the left part of its body being destroyed over an extent of three lines at the point corresponding with the aneurismal sac.

Opacity of the arachnoid coat and of the pia mater: serum between these two membranes.

Slight atrophy of the left optic nerve after the decussation.

Remarks.—Could so large an aneurismal tumor have been produced in the short space of ten weeks, especially considering that the middle arterial coat extended throughout the whole tumor and the internal, over a considerable portion? If it existed previously it is remarkable that the patient should have enjoyed robust health without either cough or expectoration.

For ten more cases of aneurism of the aorta by the author the reader may refer to the London Medical Gazette, September 5th and 12th, 1829.

FINIS.

Charles Whittingham, Tooks Court,
Chancery Lane, London.